ATLAS OF

CARDIOVASCULAR DISEASES

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CORRELATION OF CLINICAL ELECTROCARDIOGRAPHY AND CARDIAC ROENTGENOLOGY WITH CLINICAL HISTORY AND AUTOPSY FINDINGS

By

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To

MY WIFE

CLARA M KARR TREIGER M D

Whose help and encouragement have been a great stimulation
In tribute to her constant inspiration
this book is affectionately dedicated

PREFACE

A good clinical history is of paramount importance for a correct cardiac diagnosis. The analysis of clinical symptoms and signs found on physical examination must be made in terms not only of abnormal anatomy, but of physiology as well. After all it is the disturbance of the normal physiology, frequently the sequence of abnormal anatomy, which brings the patient to the clinician.

Roentgenological examination of the heart provides the clinician with data as to the gross anatomy of the heart with changes in size, shape and position of the heart and its various chambers. The measurement of size is of little value; it is of far greater importance to find out which chambers are involved in abnormal process. To obtain complete information the left and right oblique views must be taken in addition to the anterior, as the greater posterior portion of the left ventricle and also both auricles are situated posteriorly and can be visualized only in oblique views.

Electrocardiography provides the clinician with valuable information as to the physiology of the heart, especially the myocardial conduction. It records any disturbance in initiation or conduction of the impulse and any deviation in the electrical axis. By *correlating* with clinical data the clinician interprets the electrocardiographic changes in conduction in terms of abnormal anatomy and physiology as well.

Correlation of history, physical findings, roentgenogram and electrocardiogram leads to a correct understanding of the degree of anatomical and physiological changes in the heart and to a proper dose of various medications for their *correction*.

Regardless of how convincing one method of examination may be, the interpretation of it without correlation with other findings may lead to mistakes and failure of therapy.

Frequent examinations and comparative studies of roentgenograms and electrocardiograms are essential in deciding whether there is any activity of the abnormal anatomical or physiological changes found

This atlas is divided into six parts Normal Heart, Rheumatic Disease, Arteriosclerotic Heart Disease, Hypertension, Syphilitic Heart Disease, and Congenital Anomalies

A cross section of common types of heart disease with autopsy findings is presented In selection of the cases many difficulties were met, especially as to availability of autopsy, roentgenographic and electrocardiographic findings in the same case An endeavor has been made to present various cases with characteristic changes shown in the roentgenogram, electrocardiogram, pathological specimen, emphasizing their clinical values Only simple, uncomplicated cases are presented By visualizing the elementary changes in uncomplicated cases of cardiac disease, it is much easier to analyze the complicated anatomical and physiological changes of some cardiac disease with more than one etiological factor

I wish to express my thanks and grateful appreciation to Dr William G Hibbs, Medical Director, and to the members of the Medical Department of the Presbyterian Hospital for kind permission to use some of their cases

IRVING J TREIGER

CONTENTS

I

NORMAL HEART

For a clinical evaluation of abnormal anatomical and physiological changes in the heart, a knowledge of normal anatomy, as shown by the roentgenogram and physiology, especially myocardial conduction as shown by the electrocardiogram, is essential

The anterior view of a roentgenogram demonstrates the aorta pulmonary artery, and the smaller anterior portion of the left ventricle the outflow tract Both auricles and the greater portion of the left ventricle the inflow tract, are located posteriorly and visualized in the left and right oblique views

In a normally built adult, the heart is in an oblique position while in a tall thin person it is vertical and in a stout, short person horizontal In young children the heart is globular and centrally located The electrocardiogram shows variations which are normal for that particular type of heart

A normal heart is rotated by scoliosis of the spine In scoliosis with convexity of the spine to the right, the heart is rotated counterclockwise, and in the anterior view, the heart contour is similar to that of the right oblique view In scoliosis with convexity of the spine to the left the heart is rotated clockwise, and in the anterior view the heart contour is similar to that of the left oblique view

A normal heart may be displaced to the right or to the left as by pneumothorax, fluid or adhesions *Except for a shifting axis*, the electrocardiogram is normal in a rotated or displaced heart

The electrocardiogram of a normal heart shows a normal initiation and transmission of the electrical impulses a normal myocardial conduction as evidenced by normal waves (P, QRS, T) and intervals (PR QRS)

The average weight of a normal heart in an adult is 250 (female) to 300 (male) grams The thickness of the myocardium of the left ventricle is 12 to 14 millimeters and that of the right is 3 to 4 millimeters

PLATE 1 NORMAL HEART—ANTERIOR VIEW

Anterior Position—The patient's sternum is in contact with the *film cassette* or fluoroscopic screen. The patient is facing the cassette in roentgenography or the examiner in fluoroscopy. The x-ray tube is back of the patient and therefore this position is called also postero-anterior (PA).

Roentgenogram, Anterior View—In a normally built adult the heart is in oblique position.

Left cardiac border

1. Convex, upper portion—aortic knob.
2. Concave, middle portion—pulmonary artery. The pulmonary conus of the right ventricle is medially to the right and the hilum to the left. The pulmonary conus is a conelike portion of the right ventricle situated immediately *below the pulmonary valve (see Plate 64). The* hilum contains secondary branches of the pulmonary artery (see Plate 56).
3. Straight, lower portion—left ventricle, anterior part, i.e., the outflow tract of the left ventricle with blood flowing out from the apex to the aorta (see Plate 15). The greater portion of the left ventricle and both auricles are located posteriorly and are visualized in the left and right oblique views (see Plates 2 and 3).

Right cardiac border

1. Upper portion—ascending aorta.
2. Lower portion—right auricular appendage. The body of the right auricle is located posteriorly.

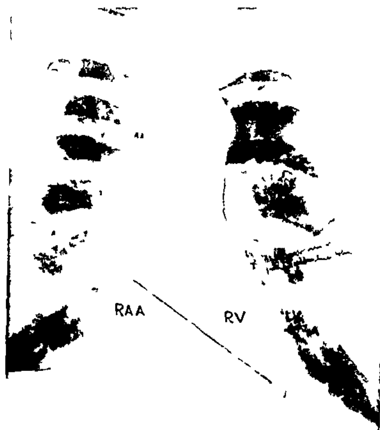
Anterior surface of the heart

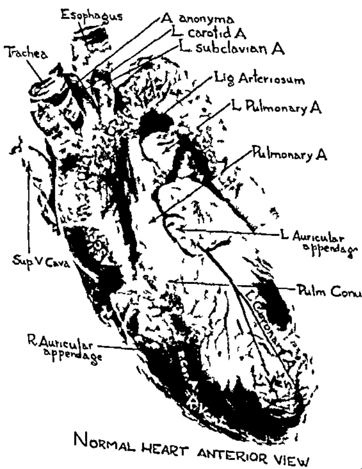
The greater central portion is the right ventricle (RV), anterior part with the pulmonary conus above and diaphragmatic surface below. The wide right lateral part is the right auricular appendage while the narrow left lateral part is the anterior portion of the left ventricle.

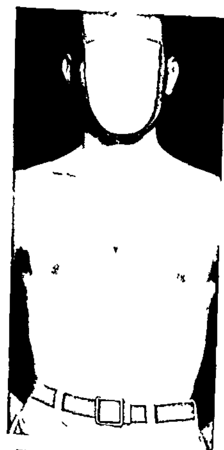
Anatomy—Correlate the anatomy and the roentgenogram. Compare with Plate 15, anterior view. The small portion of the heart below the right auricular appendage is the diaphragmatic surface of the right ventricle and is not *visible on the roentgenogram.*

Summary—The anterior view of a roentgenogram demonstrates the aorta, pulmonary artery and the smaller anterior portion of the left ventricle, the outflow tract. Both auricles and the greater portion of the left ventricle, the inflow tract, are located posteriorly and not visualized in this view.

In fluoroscopy, while the left ventricle pulsates inward, the pulmonary artery pulsates outward. The point of opposite pulsations is the upper end of the interventricular septum and the left ventricular border.







The patient is facing the cassette or the fluoroscopic scre n

PLATE 2 NORMAL HEART—LEFT OBLIQUE VIEW

Left Oblique (LO) Position.—The patient's left shoulder is *turned toward* the fluoroscopic screen or film cassette. The patient (heart) is rotated clockwise around the longitudinal axis about 50 degrees. To obtain a correct position, bisect the 90 degree angle, formed by the anterior and lateral positions, thus placing the patient at 45 degrees. Rotate the patient then slightly 5 degrees more, *in the same direction. Move the tube 2 feet closer to the* patient, thus taking the oblique views at a distance of 4 feet.

Roentgenogram, Left Oblique View.—*The spine is on the left side.* The clockwise rotation brings the left auricle and the *posterior part of the left* ventricle to the front.

Left (posterior or spinal) cardiac border

1. Upper portion—left auricle. The left bronchus and aortic arch are over the triangularly shaped, pear-shaped left auricle. The border of the left auricle adjacent to the left bronchus is a straight line.
2. Lower portion—left ventricle posterior part, inflow tract with blood flowing in from the left auricle to the apex. The normal left ventricle barely touches the spine, and the border is slightly convex.

The heart in the left oblique view is pear shaped.

The angle formed by the lower end of the left ventricular border and the diaphragm is the interventricular groove and the lower end of the interventricular septum. The upper end of the septum is the point of opposite pulsations in anterior view (see Plate 1, description).

Right (anterior or sternal) cardiac border

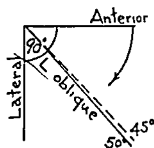
1. Upper portion—ascending aorta.
2. Middle portion—right auricular appendage.
3. Lower portion—right ventricle anterior part.

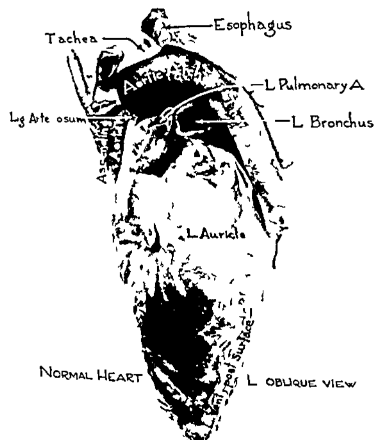
Anatomy.—The heart is rotated clockwise around the longitudinal axis. Correlate the anatomy and the roentgenogram. Note the left auricle with the left bronchus and aortic arch above and the left ventricle posterior surface, below. Compare also with Plate 15 and note the outflow and inflow tracts of the left ventricle. The tip of the clockwise rotated heart lies on *the diaphragm* and is not visible on the roentgenogram.

Summary.—The left oblique view demonstrates the arch of the aorta, the left auricle and its relation to the left bronchus and the posterior, the greater portion of the left ventricle, the inflow tract. The normal heart in the left oblique view is pear shaped.

PLATE 2 NORMAL HEART—LEFT OBLIQUE VIEW









The patient s left shoulder is turned toward film cassette or fluoroscopic screen

PLATE 3 NORMAL HEART—RIGHT OBLIQUE VIEW

Right Oblique (RO) Position.—The patient's right shoulder is turned toward the fluoroscopic screen or film cassette. The patient (*heart*) *is rotated* counterclockwise around the longitudinal axis about 50 degrees. To obtain a correct position bisect the 90 degree angle formed by the anterior and lateral positions thus placing the patient at 45 degrees. Rotate the patient then slightly, 5 degrees more, in the same direction. The x-ray tube is at a distance of 4 feet from the patient.

Roentgenogram Right Oblique View.—The spine is *on the right side*. The counterclockwise rotation brings both auricles to the front on the right border while the aortic knob, pulmonary artery, and the left ventricle on the left disappear posteriorly.

The heart in the right oblique view is triangular in shape with its base on the right side.

Right (posterior or spinal) cardiac border:

1. Upper portion—left auricle.
2. Lower portion—right auricle. The left auricle anatomically situated above the right, the septum lying horizontally between them.

Note the translucent retrocardiac space with the esophagus in it.

Left (anterior or sternal) cardiac border:

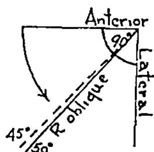
1. Upper portion—ascending aorta. Note the chimneylike appearance.
2. *Middle portion—pulmonary conus of the right ventricle.*
3. Lower portion—right ventricle, anterior part, with border close to the anterior chest wall.

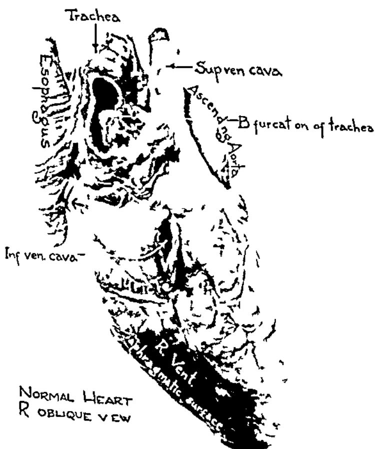
Anatomy.—The heart is rotated counterclockwise around the longitudinal axis. Correlate the anatomy and the roentgenogram. Note the close proximity of the tip of the left auricle to the straight esophagus. Note the ascending aorta, pulmonary conus of the right ventricle, and the anterior portion of the right ventricle on the left cardiac border. Compare also with Plate 5 and note the relationship of the *left auricle to the esophagus*. The tip of the counterclockwise rotated heart lies on the diaphragm and is not visible on *the roentgenogram.*

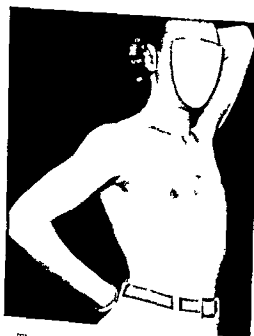
Summary.—The right oblique view demonstrates the left and right auricles and their relation to the esophagus. Due to counterclockwise rotation the aortic knob, the pulmonary artery, and the left ventricle on the left border *disappear posteriorly and are replaced by the chimneylike aorta*, the pulmonary conus of the right ventricle and the anterior portion of the right ventricle with border close to the anterior chest wall. The normal heart in the right oblique view is triangularly shaped. While taking the right oblique view, give patient barium to visualize the esophagus and its relation to the left auricle.

PLATE 3 NORMAL HEART—RIGHT OBLIQUE VIEW









The patient's right shoulder is turned toward the film cassette or fluoroscopic screen

PLATE 4 NORMAL VARIATIONS IN POSITION OF THE HEART

In a normally built adult the heart is in an oblique position see Plate 1

Fig A Vertical Heart—It appears long and narrow, the diaphragm is low, *is seen in tall, slim persons*

G F male white, aged 41 Height 5 feet, 9 inches weight 121 pounds *Functional soft systolic murmur heard in the mitral area*

In marked cases the heart appears suspended and drop shaped (drop heart) *In vertical heart the contour of the pulmonary artery may form the* larger part of the contour below the aortic knob The anatomical apex is directed medially

Electrocardiogram QRS_1 low voltage

Fig B Horizontal Heart—*The diaphragm is high as seen in short* obese persons or when the diaphragm is pushed upward by a pregnant uterus large ovarian cyst ascites etc The anatomical apex is directed laterally The left ventricular border remains straight thus differentiating it from left ventricular hypertrophy or dilatation

Electrocardiogram QRS_3 of low voltage or low notched inverted with T_3 frequently inverted also

Fig C Globular Heart—Child aged 3½ female white The heart is centrally located and its borders reach about equally far to the left and right Normal in children only

Electrocardiogram QRS_1 inverted right axis deviation as frequently seen in children

Summary—In a normally built adult the heart is in an oblique position while in a tall thin person it is vertical and in a stout short person horizontal In young children the heart is globular and centrally located

The electrocardiogram shows shifting in electrical axis which is normal for that particular type of heart In vertical heart the change in QRS as low voltage is in Lead 1 while in horizontal heart it is in Lead 3 frequently associated with inverted T_3 Inverted QRS_1 is often found normally in children especially with globular hearts

PLATE 4 NORMAL VARIATIONS IN POSITION OF THE HEART



Fig A Vertical heart The diaphragm is low



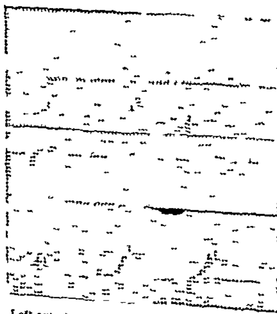
Vertical heart QRS_1 low voltage



Fig C Globular heart (child)



Fig B Horizontal heart The diaphragm is high



Left axis deviation QRS_1 diphasic QRS_3 inverted

PLATE 5 ROTATED AND DISPLACED NORMAL HEARTS

Fig A—Scoliosis with convexity of the spine to the right

The heart is rotated counterclockwise around the longitudinal axis as in the right oblique position Compare with the roentgenogram Plate 3

Note the disappearance of the aortic knob and apparent straightening of the left upper cardiac contour due to rotation The left lower cardiac border is formed not by the left ventricle but by the anterior portion of the right ventricle Note also the translucent retrocardiac space The heart is triangular in shape

Fig B—Scoliosis with convexity of the spine to the left

The heart is rotated clockwise around the longitudinal axis as in the left oblique position The round shadow on the left is that of the breast Compare with the roentgenogram Plate 2 The heart is pear shaped

Fig C—The heart is displaced to the right—acquired dextrocardia

P T female white aged 26 Far advanced tuberculous process in the *right lung pulled the heart completely into the right side of the chest* Note gas in the stomach below the left diaphragm the stomach being in normal position thus ruling out situs inversus

A normal electrocardiogram in this case ruled out a congenital dextrocardia which shows characteristic electrocardiographic changes (see Plate 62)

Summary—Scoliosis rotates the heart and changes its contour in the roentgenogram In the anterior view, the heart contour is similar to that in the left or the right oblique view A marked degree of rotation of the heart in scoliosis may cause twisting of large blood vessels with circulatory disturbance and cardiac failure in the absence of cardiac disease

A normal heart may be displaced to the right and simulate a congenital dextrocardia which may be ruled out by a normal electrocardiogram

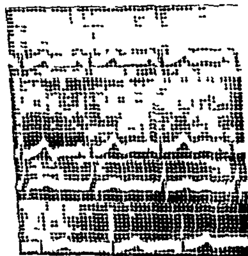
PLATE 5 ROTATED AND DISPLACED NORMAL HEARTS

Fig A Scoliosis with spine convexity to the right The heart is rotated counterclockwi as in right oblique position

Fig B Scoliosis with spine convexity to the left The heart is rotated clockwi e as in left oblique po ition



Fig C Acquired dextrocardia The heart is displaced completely to the right and obliterated by the density of the right chest



Acquired dextrocardia normal ECG

In congenital dextrocardia the heart is a mirror picture and shows characteristic ECG changes

Leads 1 and 4 completely inverted

Leads 2 and 3 replace each other (See Plate 6?)

PLATE 6 NORMAL ELECTROCARDIOGRAM

The *electrocardiographic* record is produced by the effect of the action current of the heart which is led off from various parts of the body

Lead 1 Right arm to left arm RA to LA

Lead 2 Right arm to left leg RA to LL

Lead 3 Left arm to left leg LA to LL

Lead 4 Precordium to left leg LL wire to precordium LA wire to LL

The precordial, chest foot (CF) lead is placed on the intersection of the midclavicular line and fifth interspace so called Position 4 on the chest (CF4 lead) Recently the use of two additional precordial leads in other positions on the chest was adopted as routine with a total number of six precordial leads *in special cases The three or more precordial leads give additional informa*tion as to the activity in the ventricular myocardium, anterior portion

The horizontal and vertical lines on the electrocardiogram are for measurements of rate PR and QRS intervals and voltage of P QRS and T The horizontal measurements are for time One small square is equivalent to 0 04 second, while one large square is equivalent to 0 2 second The vertical measurements are for voltage (height or depth) One small square is equivalent to 0 1 millivolt and is 1 millimeter in height

The rate is normally 60 to 100 beats per minute The usual length of the mounted electrocardiographic record each lead is slightly over 6 inches or 30 large squares or 6 seconds in time The number of cycles (the space between two QRS complexes) in a 6 inch strip multiplied by ten gives the approximate heart rate

The PR interval is the conduction time from the sinus node to the ventricles The upper limit of normal in adults is 0 20 second, in children it is *0 16* second

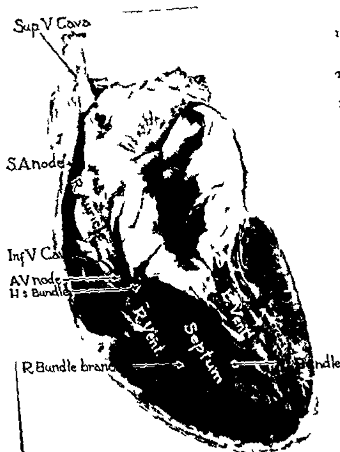
The QRS interval is the intraventricular conduction time and is measured at the base of the QRS The upper limit of *normal in adults is 0 10 second* in children it is 0 09 second

The normal rhythm is the so *called* sinus rhythm with *normal position* and contour of P in reference to the QRS

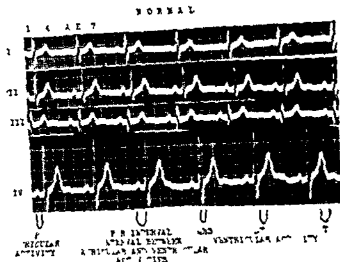
A normal electrical axis in the electrocardiogram is shown by an upright QRS in Leads 1 2, 3

Summary The electrocardiogram gives us valuable information as to the physiology of the heart the rate rhythm conduction in the myocardium of the auricles and ventricles *and conduction systems This information* correlated with clinical and roentgenologic findings is of great value as to estimating the anatomical changes in the heart if any are present

PLATE 6 THE CONDUCTION SYSTEM OF THE HEART AND NORMAL ELECTROCARDIOGRAM



AV Conduction System





Sinoauricular (S A) and auriculoventricular (A V) conduction systems

A plexiform network of neuromuscular tissue spread through the walls of the heart. Sinoauricular node situated in the wall of the right auricle near the orifice of superior vena cava. Auriculoventricular node situated in the septal wall of the right auricle immediately above the opening of coronary sinus.

The impulse starts at the sinoauricular node (the pacemaker) and is transmitted to the myocardium of both auricles. P deflection in ECG is due to auricular activity. From the auricle the impulse is transmitted to the auriculoventricular node. From here the conduction fibers as the bundle of His run forward on the septum.

PR interval in ECG (measured from the beginning of the P to the beginning of the QRS) indicates the time required for the transmission of the impulse from the sino auricular node to the ventricles.

The His bundle separates into right and left branches to spread out over the ventricles.

QRS and T deflections in ECG are due to ventricular activity.

PLATE 6 NORMAL ELECTROCARDIOGRAM

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Lead 1 Right arm to left arm RA to LA

Lead 2 Right arm to left leg RA to LL

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Lead 4 Precordium to left leg LL wire to precordium, LA wire to LL

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Summary The electrocardiogram gives us valuable information as to the physiology of the heart the rate rhythm conduction *in the myocardium of* the auricles and ventricles and conduction systems This information correlated with clinical and roentgenologic findings is of great value as to estimating the *anatomical changes* in the heart if any are present

trophy sets in early, as well as physiological adjustment of the blood pressure. The more pronounced is the regurgitation, the more there is hypertrophy of the left ventricle as seen on the roentgenogram. In myocarditis and pericarditis, electrocardiographic changes may completely disappear, or if the scar tissue and fibrosis interfere with conduction, permanent electrocardiographic changes may remain as residue of a healed condition. At this stage a murmur as evidence of a valvular lesion, auricular or ventricular hypertrophy, electrocardiographic and roentgenographic changes are signs of an inactive, healed and well compensated process, and the patient does not need any treatment for his condition.

If for any reason a breakdown of compensation occurs, a cardiac failure with failure of the function of the ventricular myocardium sets in and the failing heart must be helped by the physician to carry on the required physiological processes or a complete breakdown will lead to death.

At this stage of breakdown of compensation the physician must evaluate the extent of anatomical and physiological impairments in the heart; he must correlate the electrocardiographic and roentgenographic findings, as, for instance, the extent of auricular or ventricular hypertrophy with clinical signs of left or right ventricular failures. The knowledge of the degree of cardiac damage and its adjustment by the heart is most helpful to the physician as to the proper dose of the medication to be used.

This introduction to rheumatic heart disease would be incomplete if we failed to mention bacterial endocarditis as a complication of a healed process, of which more will be said in a special chapter assigned to it.

1. MITRAL STENOSIS

The function of a normal mitral valve is (1) to close during the ventricular systole in order to prevent the blood from flowing back from the left ventricle into the left auricle, the closure of the valve causes the normal first mitral heart sound which is therefore systolic in time; (2) to open during the ventricular diastole in order to let the blood flow from the left auricle into the left ventricle.

II

RHEUMATIC HEART DISEASE

In dealing with rheumatic heart disease the examining physician must ascertain first whether the condition is active or inactive. The terms acute, subacute, and chronic, as used by the pathologist when handling pathologic specimens, should not be used by clinicians dealing with human beings with various normal and abnormal, active and inactive, physiological processes. Fever, leucocytosis, and a high *sedimentation rate are examples of some of the general signs of activity*, while the appearance of a murmur, pericardial rub, or electro*cardiographic changes are some of the topical, local signs of activity in* the heart.

Active rheumatic fever may affect the endocardium with special predilection for valvulitis with mitral or aortic insufficiency or both and left auriculitis.

The myocardium may be affected primarily or secondarily, by extension from the endocardium. The inflammation may involve the ventricular wall or the interventricular septum or both.

The pericardium, the same as the myocardium, may be affected primarily or secondarily from extension from the endocardium and myocardium, causing carditis or pancarditis. *Pericardial rub*, roentgenograms, and electrocardiographic changes, as low voltage, inversion of all the T waves, are some of the important diagnostic signs. The electrocardiographic abnormalities, if present, are valuable clues in doubtful or unsuspected cases of pericarditis.

The stage of activity and healing is followed by an inactive stage of a healed process with anatomical and physiological adjustments. Following mitral insufficiency, a fibrosis of the valve sets in, causing a mitral stenosis which is indication of a healed condition, with moderate or marked hypertrophy of the left auricle to compensate for it. The more pronounced is the stenosis, the more there is hypertrophy and enlargement of the left auricle as shown on the roentgenogram. In aortic insufficiency *the aortic valve has considerably less tendency to* fibrosis and stenosis than the mitral valve. Left ventricular hyper

deviation The extent of the left auricular enlargement and cardiac rotation caused by it, and shown on the roentgenogram is in direct proportion to the degree of mitral stenosis

When fibrosis and stenosis of the mitral valve is associated with auriculitis, this may cause abnormal auricular myocardial conduction, which will manifest itself in the electrocardiogram by deformed P wide, notched high or low, diphasic or inverted The anatomical changes in the left auricle may cause increased irritability and formation of auricular premature systoles, paroxysmal auricular tachycardia (which is a succession of numerous auricular premature systoles), or, as frequently is the case, auricular fibrillation with abnormal initiation and conduction of auricular impulses

Auricular fibrillation per se is not a sign of left or right ventricular failure It is only an abnormal auricular rhythm and may be associated with a ventricular rate, which is slow, normal or fast The fast rate unless it is paroxysmal, is evidence of ventricular failure and must be treated accordingly with digitalis Clinically, there may be dyspnea with or without edema and cyanosis, while the electrocardiogram may show low voltage of QRS and P's as evidence of myocardial insufficiency While auricular fibrillation in mitral stenosis may be frequently a precursor of cardiac failure there are a great number of cases in which it may persist for many years without any evidence of failure

As long as the mitral stenosis is well adjusted or compensated by hypertrophy and dilatation of the left auricle and hypertrophy of the right ventricle, the mitral stenosis, if uncomplicated is an anatomical residue of an old healed rheumatic heart disease, well adjusted and therefore as such does not *require* any treatment When a break down of compensatory mechanisms occurs, cardiac failure with impaired function of the right ventricle develops as cyanosis, peripheral edema, ascites enlargement of the liver, hydrothorax, complicated by thrombo embolic processes which may cause the patient's death if the events cannot be controlled in time

The anatomical changes in mitral valvulitis manifest themselves clinically by appearance of a murmur. In the active stage, the mitral valve is impaired to such a degree that it cannot close sufficiently during the ventricular systole, and blood will regurgitate into the left auricle, the murmur formed will be systolic in time, occurring during ventricular systole. In the inactive stage the fibrosis and mitral stenosis will interfere with opening of the valve and normal blood flow during the ventricular diastole, and therefore the murmur will be diastolic in time. This diastolic murmur depends in its formation on auricular systole (P wave in the electrocardiogram), which occurs in the late phase of ventricular diastole and shortly preceding the ventricular systole (QRS, ST, T in the electrocardiogram), the murmur in mitral stenosis is therefore late diastolic or so called presystolic. When mitral stenosis is complicated by auricular fibrillation, the normal mechanism of auricular systole (P wave in the electrocardiogram) is gone, and therefore the murmur cannot be presystolic any more, it becomes a mid diastolic murmur, occupying the mid diastolic phase of diastole. The presystolic mitral murmur is normally a rumble of the same intensity. The rumble is usually heard in the localized mitral area, is not transmitted, and may be influenced by a change in the patient's position and by exercise.

As the apex is displaced to the left and down on account of the rotation of the heart, the rumble is usually heard in the sixth interspace near the anterior axillary line. The blood, while flowing from the left auricle into the left ventricle, meets with a considerable resistance of the stenotic valve, therefore the diastolic murmur is of rumble quality. This is in contradistinction to a soft diastolic murmur of aortic insufficiency when blood regurgitates freely from the aorta into the left ventricle and does not meet any resistance in the valve during its flow.

Anatomical changes in the mitral valve, fibrosis and stenosis, are soon adjusted or compensated by hypertrophy and dilatation of the left auricle and later by hypertrophy of the right ventricle. The enlargement of the left auricle progresses gradually posteriorly toward the esophagus compressing it, and to the right and anteriorly, thus rotating the heart counterclockwise on its vertical axis. These findings can be well shown on the roentgenogram and in some cases the electrocardiogram, by high peaked P's especially P and right axis

PLATE 7 MITRAL STENOSIS WITH ADVANCED CARDIAC FAILURE



Heart *in situ* Heart rotated counter clockwise by the enlarged *left auricle*

Mitral valve as seen from above Marked mitral stenosis Marked dilatation and hypertrophy of the left auricle

The right auricle and auricular appendage are enlarged due *to* thrombosis and cardiac failure and pushed out on the right side by the hypertrophied and enlarged right ventricle

Due to rotation *the* pulmonary conus of the right ventricle bulges on the left upper border the hypertrophied anterior portion of the right ventricle forms the left lower cardiac border

Due to *counterclockwise* rotation the aortic knob *pulmonary artery* and anterior portion of the left *ventricle* with the apex are displaced *posteriorly*

The left auricle is *markedly* distended The *lining* is gray and opaque The mitral valve is *markedly altered* Instead of the usual *opening* there is a linear slit having a maximum diameter *of* about 12 to 15 *mm* and when stretched it is 2 to 4 mm wide The two leaflets are adherent at *their* adjoining margins to produce a complete singular leaflet the anterior leaflet has a *maximum* width of 2 8 cm the posterior 1 cm Where the two leaflets join posteriorly the *auricular surface* is jagged and the crevice is filled with reddish brown material resembling clotted blood

PLATE 7 MITRAL STENOSIS WITH ADVANCED CARDIAC FAILURE

J. C., male, aged 54. Chorea in childhood. In the past ten years a gradually progressive dyspnea developed, recently so severe that he needed three or four pillows at night. The symptoms progressed until the patient was weak and emaciated, and apparently in the last stage of cardiac failure.

The physical examination revealed a pale, dehydrated, tired male, acutely ill, sitting up in bed and breathing with difficulty. Pulse 92, temperature *98, respirations, 24. Heart: the apex beat diffuse and distant; a diastolic* murmur heard at the apex; heart action irregular due to auricular fibrillation. Lungs: right hydrothorax. The liver palpable five fingerbreadths below *the right costal border, the edge sharp*; ascites with pitting edema of both feet. Moderate amount of albumin in the urine. Two days after admission he was having difficulty with speech and was disoriented. Three days later he was having involuntary urination and was comatose, cyanotic, and had increased respirations with gradual failure of the pulse. He died six days after admission.

Autopsy.—Chronic indurative mitral, aortic and tricuspid endocarditis; mitral stenosis; marked dilatation and hypertrophy of the left auricle and right ventricle; dilatation of the tricuspid *orifice; thrombosis of the right auricular* appendage.

Generalized passive hyperemia; chronic passive hyperemia of the lungs; right sanguinohydrothorax; thromboembolism of the branches of the right pulmonary artery; slight icterus.

Marked sclerosis of the pulmonary artery, thoracic and abdominal aorta; coronary arteries patent.

Summary.—A case of mitral stenosis with advanced cardiac failure is presented to demonstrate the heart in situ and the stenotic mitral valve.

PLATE 7 MITRAL STENOSIS WITH ADVANCED CARDIAC FAILURE



Heart in situ. Heart rotated counterclockwise by the enlarged left auricle.

Mitral valve as seen from above. Marked mitral stenosis. Marked dilatation and hypertrophy of the left auricle.

The right auricle and auricular appendage are enlarged due to thrombosis and cardiac failure and pushed out on the right side by the hypertrophied and enlarged right ventricle.

Due to rotation the pulmonary conus of the right ventricle bulges on the left upper border; the hypertrophied anterior portion of the right ventricle forms the left lower cardiac border.

Due to counterclockwise rotation the aortic knob, pulmonary artery and anterior portion of the left ventricle with the apex are displaced posteriorly.

The left auricle is markedly distended. The lining is gray and opaque. The mitral valve is markedly altered. Instead of the usual opening there is a linear slit having a maximum diameter of about 1[illegible] to 1[illegible] mm and when stretched it is [illegible] to 4 mm wide. The two leaflets are adherent at their adjoining margins to produce a complete singular leaflet; the anterior leaflet has a maximum width of 1.8 cm, the posterior 1 cm. Where the two leaflets join posteriorly the auricular surface is jagged and the crevice is filled with reddish brown material resembling clotted blood.

PLATE 8 MITRAL STENOSIS

E. F., female, white, aged 47, died at home in June, 1944.

Longitudinal section of the heart, rotated counterclockwise on its vertical axis by an enlarged left auricle.

The stenosis of the mitral valve is of marked degree so that it is difficult to pass the little finger through it. This stenosis is characterized by thickening of the leaflets, obliteration of the commissures, thickening of the chordae tendineae and retraction especially of the posterior leaflet, all characteristics of chronic indurative mitral endocarditis such as is observed in rheumatic heart disease. The wall of the left auricle is 3 to 5 millimeters thick and dense from proliferation of gray fibrous tissue. It is increased in size from dilatation and its lumen is partially filled with masses of mottled tan and brownish tan thrombi resembling those of a mixed thrombus.

The longitudinal section of the heart shows that the left auricular enlargement extends posteriorly toward the esophagus, compressing it, and to the right and anteriorly, thus rotating the heart counterclockwise on its vertical axis. The enlargement extends to the left only as far as the left auricular appendage, and therefore cannot be visualized in the roentgenogram along the left cardiac border.

Summary.—A case of mitral stenosis is presented to demonstrate the close similarity of cardiac outlines in the roentgenogram and in the heart specimen. The longitudinal section demonstrates the extent of the left auricular enlargement posteriorly, compressing the esophagus, and to the right and anteriorly, rotating the heart counterclockwise on its vertical axis.

PLATE 8 MITRAL STENOSIS



A Heart rotated counterclockwise as in right oblique position The aortic knob is hardly visible the pulmonary conus of the right ventricle is below it and the anterior part of the right ventricle forms the lower portion of the left cardiac border The apex is rounded and displaced to left and down

B Note close similarity of cardiac outlines here and in roentgenogram



Longitudinal section of the counterclockwise rotated heart as in right oblique position Esophagus is indented by an enlarged left auricle

PLATE 9 INACTIVE MITRAL STENOSIS

L M, female white, aged 32

Acute rheumatic fever at 15 years of age three months in bed Well and active since, except for slight dyspnea on exertion A presystolic rumble heard in mitral area not transmitted Blood pressure, 100/70 No physical findings of aortic or other valve involvement Roentgenograms show characteristic changes due to enlarged left auricle

Anterior View—Compare with normal, Plate 1

The heart has slightly rotated counterclockwise on its vertical axis and therefore the left cardiac border has partially disappeared posteriorly The aortic knob is hardly visible The pulmonary artery contour, which is normally concave, has due to rotation been replaced by the pulmonary conus of the right ventricle, which lies medially to it and the left mid cardiac border is therefore straight The anterior part of the right ventricle forms the lower portion of the left cardiac border The apex is rounded

Left Oblique View—Compare with normal Plate 2

Normally the border of the left auricle adjacent to the left bronchus is a straight line and the left auricle is of a triangular peaked shape *In here, the border of the left auricle adjacent to the left bronchus is curved* due to enlargement, thus deforming the normal triangular shape of the left auricle The left bronchus is elevated and compressed by the enlarged left auricle

Right Oblique View—Compare with normal Plate 3

The normally straight esophagus is indented and curved by the enlarged left auricle

Summary — A case of inactive mitral stenosis is presented to demonstrate characteristic roentgenographic findings straightening of the left mid cardiac contour elevation and compression of the left bronchus indentation and curving of the esophagus

PLATE 9 INACTIVE MITRAL STENOSIS



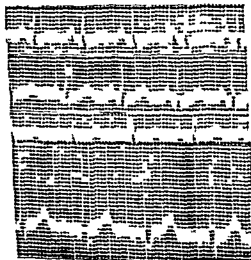
Pulmonary contour is straight (normally concave) due to slight counterclockwise rotation of the heart by the enlarged left auricle



Left oblique view Left bronchus elevated and compressed by the enlarged left auricle The border of the left auricle is curved



Right oblique view Barium filled esophagus indented and curved by the enlarged left auricle Note pulmonary conus bulge on the left border



P1 2 peaked relatively high auricular hypertrophy Normal electrical axis

PLATE 10 MITRAL STENOSIS OF MARKED DEGREE

V P, male, white, aged 28

Rheumatic fever followed by heart disease, twelve years previously (1931), at the age of 16 At the time the roentgenogram and electrocardiogram were taken (April 27, 1943), the patient's complaints were palpitation insomnia, slight gastrointestinal distress Heart apex displaced to left and down due to cardiac rotation diastolic murmur in the mitral area auricular fibrillation In September, 1945 the patient developed bacterial endocarditis for which large doses of penicillin were given Fever chills, and later petechiae and nodules on extremities A month later, cerebral embolism, unconsciousness, right hemiplegia, with motor aphasia, facial paralysis, and impaired vision in the right eye In November, he developed acute cardiac failure and was not expected to live In January, 1946, he left the hospital markedly improved and at present (December, 1946) is doing fairly well

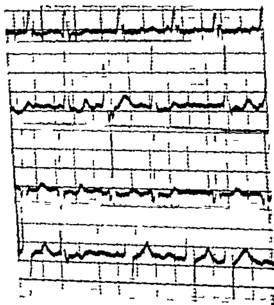
Roentgenogram—The left auricle is markedly enlarged and appears in the *anterior view as an area of increased density in the middle portion* of the right cardiac contour above it is the ascending aorta, below the right auricle The left border of the enlarged left auricle would be, if visible, *slightly to the left of the sternum, but would not extend all the way to the* left cardiac border (see Plate 8) The enlargement of the left auricle while extending to the right and anteriorly causes rotation of the heart counter clockwise *on its vertical axis as in the right oblique position (see* Plate 3) The aortic knob pulmonary artery left ventricular border and apex disappear posteriorly due to rotation The present left cardiac contour is formed by *straight aorta, prominent pulmonary conus of the right* ventricle and the hypertrophied right ventricle anterior portion

Summary—A case of marked mitral stenosis is presented to demonstrate on the roentgenogram the enlarged left auricle appearing on the right cardiac contour and the counterclockwise rotation of the heart

PLATE 10 MITRAL STENOSIS OF MARKED DEGREE



Heart markedly rotated counterclockwise as in right oblique position
Enlarged left auricle seen on the right side
Due to rotation aortic knob left ventricular border apex di appeared posteriorly and replaced by straight aorta pulmonary conus and anterior portion of the right ventricle



Auricular fibrillation

PLATE 11 ELECTROCARDIOGRAM IN MITRAL STENOSIS AND AURICULAR FIBRILLATION

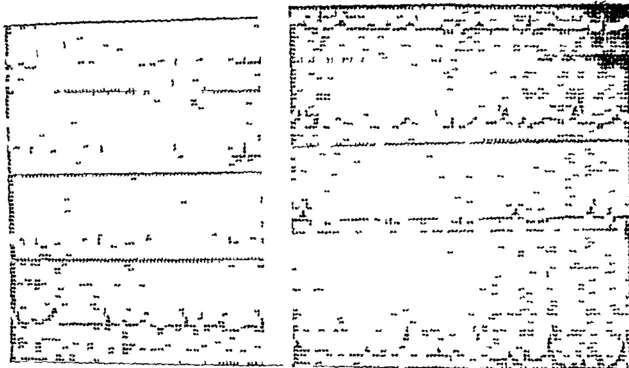
F T, male, white, aged 21 Admitted on July 28, 1941, died on Oct 26 1941, of advanced heart failure

Chorea at 11 years of age followed by heart disease Three years later patient developed dyspnea on exertion palpitation, precordial pain Examination on April 25, 1935 *Heart regular, enlarged to the left the left heart border in midaxillary line in the sixth interspace* diastolic thrill over the apex presystolic rumble heard in the same area Blood pressure 140/58 *Electrocardiogram* P *notched* wide or *high* suggesting auricular hypertrophy and abnormal auricular conduction (auriculitis) also right axis deviation indicating right ventricular hypertrophy The clinical and electrocardiographic findings were about the same as above on March 11 1937 and May 1 1939 The patient returned on Oct 11 1939 with auricular fibrillation mitral diastolic murmur, and advanced heart failure Admitted to the hospital with right hydrothorax markedly enlarged liver ascites, edema of lower extremities died three months later No autopsy

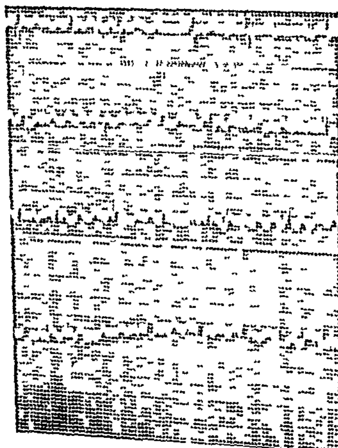
The presence of auriculitis may be revealed in the electrocardiogram by changes in P waves due to impaired auricular myocardial conduction or by abnormal initiation and conduction of impulses as auricular premature systoles paroxysmal auricular tachycardia (which is a succession of numerous auricular premature systoles) auricular fibrillation or flutter suggesting *irritable auricle*

Summary—A *case of mitral stenosis with characteristic electrocardiograms* is presented Etiological diagnosis Rheumatic fever (chorea) in 1931 Anatomical diagnosis Mitral stenosis (valvulitis) and auriculitis Physiological diagnosis Mild cardiac insufficiency with impaired auricular *myocardial conduction in 1934 followed by* auricular fibrillation and advanced cardiac insufficiency in 1939 and death two years later

PLATE 11 ELECTROCARDIOGRAM IN MITRAL STENOSIS AND AURICULAR FIBRILLATION



P notched wide or high— auricular hypertrophy with abnormal auricular conduction
Right axis deviation



Auricular fibrillation Right axis deviation

2 AORTIC INSUFFICIENCY

The function of a normal aortic valve is (1) to close during the ventricular diastole in order to prevent the blood from flowing back from the aorta into the left ventricle, the valve closes by the pressure of the blood column in the aorta, and the closure causes the normal second aortic sound, which is therefore diastolic in time, (2) *to open during the ventricular systole* in order to let the blood flow from the left ventricle into the aorta

The anatomical changes in aortic valvulitis manifest themselves clinically by *appearance of a murmur* *This murmur will* be diastolic in time, as the insufficient valve cannot close well enough during the ventricular diastole in order to prevent the blood from regurgitating from the aorta into the left ventricle The diastolic murmur is usually soft and frequently heard best in the left third interspace near the sternal border The aortic soft diastolic murmur is often accompanied by a functional soft diastolic murmur in the mitral area, Austin Flint murmur, not a rumble as in mitral stenosis At times it may be difficult to decide if the mitral diastolic murmur is not caused by an *associated mitral stenosis* *In that case, to the* characteristic roentgenographic changes of left ventricular hypertrophy due to aortic insufficiency and shown in *the* anterior and left oblique views may be added hypertrophy of the left auricle, which indents and curves the esophagus in the right oblique view The roentgenogram, electrocardiogram, and blood pressure changes may well be correlated with *clinical findings to decide whether the aortic insufficiency or mitral* stenosis, if both are present, is predominant

Besides the aortic diastolic murmur, aortic insufficiency has a characteristic pulse sharply rising sharply peaked *and abruptly* dropping

Anatomical changes in aortic insufficiency are soon adjusted or *compensated by physiological* adjustment of the blood pressure and anatomical hypertrophy of the left ventricle The systolic pressure, i e, *the power of the heart,* is *slightly elevated in order* to propel into

the aorta during ventricular systole the normal volume of blood, which came from the left auricle plus the regurgitated blood during the preceding diastole The diastolic pressure, i e, the peripheral resistance is usually markedly depressed, not so much due to regurgitation, as to a marked vasodilatation frequently associated with aortic insufficiency The blood pressure change is so striking that when found, it should lead one to suspect aortic insufficiency

The left ventricular hypertrophy can well be shown on the roentgenograms, anterior and left oblique views (see Plate 12) The hypertrophy is in direct proportion to the insufficiency of the valve, the more the valve is insufficient, the more blood is regurgitated and the more hypertrophy is caused The average thickness of the left ventricular myocardium is about 12 to 14 millimeters The myocardium may increase in size to a certain degree, perhaps to about twice the average size, after which the left ventricle begins to dilate, and fails The roentgenogram in dilatation will show that the left cardiac border is not only rounded but extends far out to the left in the anterior view and markedly overlaps the spine in the left oblique view

The electrocardiogram in addition to the roentgenogram may furnish evidence of left ventricular hypertrophy left axis deviation or left axis deviation with high QRS or left ventricular strain, i e, left axis deviation with high QRS, depressed ST_1, and deeply inverted T_1 and often T_4

As long as the aortic insufficiency is well adjusted or compensated by hypertrophy of the left ventricle and blood pressure changes, the aortic insufficiency, if uncomplicated, is an anatomical residue of an old, healed rheumatic heart disease, well adjusted, and therefore as such does not require any treatment When a breakdown of compensatory mechanism occurs, dilatation and cardiac failure with impaired function of the left ventricle develop as dyspnea on exertion, orthopnea, and later attacks of paroxysmal nocturnal dyspnea, and if not controlled in time may soon lead to right ventricular failure and death

PLATE 12 INACTIVE RHEUMATIC AORTIC INSUFFICIENCY AND MITRAL STENOSIS

A. B., male, white, aged 23.

Streptococcus sore throat at age 15, followed later by the appearance of heart murmurs. Patient has been well and active since. Mild soft diastolic murmur at the third left interspace near sternal border, mild presystolic rumble at the mitral area *not transmitted. Pulsating and dilated blood* vessels of the neck and extremities. Corrigan pulse. Blood pressure 140/50.

Roentgenograms show evidence of mild hypertrophy of the left ventricle *due to aortic insufficiency and of the left auricle due to mitral* stenosis. The hypertrophy of the left ventricle is the predominant feature in the roentgenogram and correlates well with predominance of aortic *insufficiency with pulse and blood pressure changes clinically. The hyper*trophy involves the inflow and outflow tracts of the left ventricle (see Plate 15 for illustration of both tracts). The inflow tract is the posterior portion of the left ventricle with blood flowing in from the left auricle to the apex of the left ventricle. The outflow tract is the anterior portion of the left ventricle with blood flowing out from the apex of the left ventricle into the aorta.

Anterior View.—Compare with normal Plate 1.

The left ventricle outflow tract is elongated; the left cardiac border instead of being straight is rounded and extends straight down below the diaphragm.

Left Oblique View.—Compare with normal Plate 2.

The left ventricle inflow tract is hypertrophied; the left cardiac border near the spine instead of being slightly curved as a bow is rounded and *the heart is therefore globular in shape* instead of being pear shaped normally.

The normal electrocardiogram in this case suggests absence of myocardial involvement.

Summary.—A case of inactive rheumatic aortic insufficiency and mitral stenosis is presented to demonstrate characteristic roentgenographic findings of left ventricular hypertrophy.

PLATE 12 INACTIVE RHEUMATIC AORTIC INSUFFICIENCY AND MITRAL STENOSIS



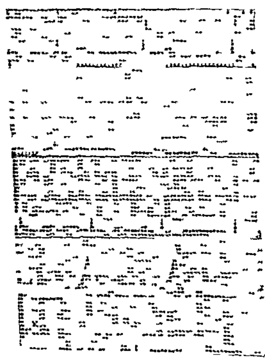
Anterior view aortic insufficiency Left ventricle (out flow tract) elongated Left cardiac contour rounded and extends straight down below the diaphragm



Left oblique view aortic insufficiency Hypertrophy of the left ventricle posteriorly (inflow tract) Increased rounding of the left cardiac contour the heart is globular in shape



Right oblique view mitral stenosis Prominent pulmonary conus on the left border Enlarged left auricle on the right border



Normal

PLATE 13 RHEUMATIC CARDITIS WITH AORTIC INSUFFICIENCY AND CARDIAC FAILURE

C B, male, white, aged 16 Last admitted on Oct 19, 1945, died on Oct 23, 1945

Acute rheumatic fever at 6½ years of age, for which he was hospitalized for a short time Recrudescence of rheumatic activity three months later, with three months' hospitalization At that time the sedimentation rate was 29 mm in one hour, pistol shot femoral pulse, loud diastolic murmur over the right base and fourth left interspace near sternum, diagnosed as aortic regurgitation In July, 1942 at the age of 12½ years the patient entered the hospital with severe abdominal pain and fever and suspected appendicitis, but later it was interpreted as being due to an acute pericarditis with effusion Patient was acutely ill for weeks The course during the next three years was fair, up until the last hospital admission On admission the apical beat was diffuse and prominent to palpation, the heart markedly enlarged the left heart border reaching the midaxillary line there were loud double murmurs in aortic and mitral areas, a Corrigan pulse blood pressure, 130/20 The liver extended three fingerbreadths down and was tender to palpation Edema of the legs ankles, and feet

Electrocardiograms A and B were practically within normal limits thus ruling out any myocardial involvement The roentgenogram at that time showed rounding of the left cardiac border due to left ventricular hypertrophy as seen in aortic insufficiency

Electrocardiogram C, taken during recovery from pericarditis $T_{1, 2, 3}$ inverted probably due to the pericarditis

Electrocardiogram D, taken less than a month after (C), showed a normal upright $T_{1, 2}$

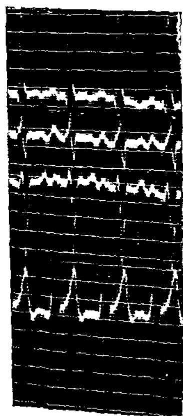
Electrocardiogram E taken four days before death showed a marked left axis deviation, with high, wide slurred QRS T_1 inverted and in correlation with the X ray and clinical findings was interpreted as left ventricular strain

Roentgenogram—The left cardiac border was not only markedly rounded as in hypertrophy, but was far out to the left near the axillary line due to marked hypertrophy and dilatation of the left ventricle Compare with Plate 12 anterior view, showing left ventricular hypertrophy only

PLATE 13 RHEUMATIC CARDITIS WITH AORTIC INSUFFICIENCY AND CARDIAC FAILURE

A 3/15/ 8 | *B* 8/6/40 | *C* 11/10/4[illegible] | *D* 1[illegible]/4/[illegible]

E 10/19/45 Aged 16



Left [illegible]

10/19/45

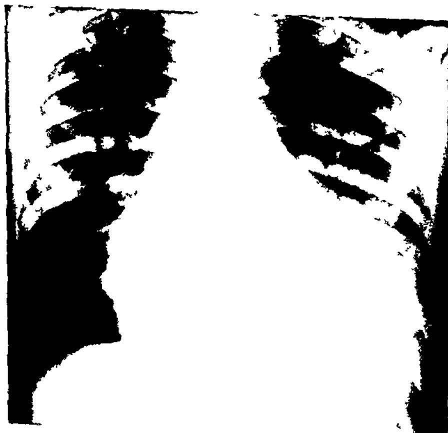


PLATE 14 RHEUMATIC CARDITIS WITH AORTIC INSUFFICIENCY, AND CARDIAC FAILURE (CONTINUED)

C B, male, white, aged 16

Autopsy—Chronic aortic endocarditis, marked aortic insufficiency marked cardiac hypertrophy old adherent fibrous pericarditis The heart fills up to one half of the thoracic cavity and weighs 1 575 grams

Chronic passive venous congestion, chronic passive hyperemia of the liver (*nutmeg*) *ascites*, *massive edema* of the legs ankles, and feet *Broncho* pneumonia, compression atelectasis of the left lung by the enlarged heart

Summary—*A case of rheumatic aortic insufficiency with advanced cardiac* failure is presented The anatomical changes in aortic valvulitis manifest themselves clinically by appearance of aortic diastolic murmur and were *soon adjusted or compensated by physiological adjustment of the blood pres*sure (130/50) and anatomical hypertrophy of the left ventricle After a period of six years of relative well being the patient developed acute pericarditis with effusion and was acutely *ill* for weeks The patient s course during the next three years was fair up until his last hospital admission with advanced left and right ventricular failure, and he died four days later Autopsy revealed a cor bovinum the heart weighing 1 575 grams while a normal heart would weigh about 250 grams

The marked aortic insufficiency caused a *marked hypertrophy of the left* ventricle which reached extreme limits and was followed by a left and later by a right ventricular dilatation and failure dyspnea cyanosis, edema of the legs ascites swelling of the liver In this case the cardiac failure and death were unavoidable results of a complete breakdown of physiological processes due to a marked aortic insufficiency which could not be well compensated for a long period of time even by the marked left ventricular hypertrophy Acute pericarditis with effusion was probably a precipitating factor in this physiological breakdown

PLATE 14 RHEUMATIC CARDITIS WITH AORTIC INSUFFICIENCY AND CARDIAC FAILURE (CONTINUED)





Cor bovinum (1 5 ? grams) Aortic insufficiency hypertrophy and dilatation of the left ventricle myocarditis adhesive pericarditis

PLATE 14 RHEUMATIC CARDITIS WITH AORTIC INSUFFICIENCY, AND CARDIAC FAILURE (CONTINUED)

C B, male, white, aged 16

Autopsy.—Chronic aortic endocarditis, marked aortic insufficiency, marked cardiac hypertrophy, old adherent fibrous pericarditis. The heart fills up to one half of the thoracic cavity and weighs 1575 grams.

Chronic passive venous congestion, chronic passive hyperemia of the liver (nutmeg), ascites, massive edema of the legs, ankles and feet. Bronchopneumonia, compression atelectasis of the left lung by the enlarged heart.

Summary.—A case of rheumatic aortic insufficiency with advanced cardiac failure is presented. The anatomical changes in aortic valvulitis manifest themselves clinically by appearance of aortic diastolic murmur, and were soon adjusted or compensated by physiological adjustment of the blood pressure (130/50) and anatomical hypertrophy of the left ventricle. After a period of six years of relative well being the patient developed acute pericarditis with effusion and was acutely ill for weeks. The patient's course during the next three years was fair up until his last hospital admission with advanced left and right ventricular failure, and he died four days later. Autopsy revealed a cor bovinum, the heart weighing 1575 grams, while a normal heart would weigh about 250 grams.

The marked aortic insufficiency caused a marked hypertrophy of the left ventricle which reached extreme limits and was followed by a left and later by a right ventricular dilatation and failure: dyspnea, cyanosis, edema of the legs, ascites, swelling of the liver. In this case the cardiac failure and death were unavoidable results of a complete breakdown of physiological processes due to a marked aortic insufficiency which could not be well compensated for a long period of time even by the marked left ventricular hypertrophy. Acute pericarditis with effusion was probably a precipitating factor in this physiological breakdown.

PLATE 14 RHEUMATIC CARDITIS WITH AORTIC INSUFFICIENCY AND CARDIAC FAILURE (CONTINUED)





Cor bovinum (1 5,6 grams) Aortic insufficiency hypertrophy and dilatation of the left ventricle myocarditis adhesive pericarditis

PLATE 15 INACTIVE MITRAL AND AORTIC STENOSIS

D S, male, white, dentist, aged 73

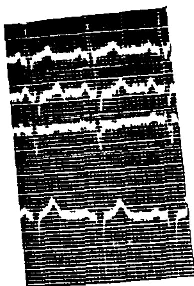
Admitted on June 6, 1944, for prostatectomy, performed four days later Died on June 19, 1944, of aspiration pneumonia

The heart weighed with aortic arch, pulmonary artery, and its branches hili of the lungs esophagus and greater part of the thoracic aorta 1 050 grams It is estimated that the heart itself without these other structures probably weighed 600 grams or more

Longitudinal section of the heart rotated clockwise and placed in left oblique position, the same as in left oblique view on the roentgenogram (see Plate 2) Pathological changes in various chambers of the heart are all shown in this one section mitral stenosis with dilated left auricle and hypertrophied *right ventricle (4 to 10 mm normally, 3 to 4 mm), also aortic stenosis with* hypertrophied left ventricle (20 mm , normally 12 to 14 mm) Both inflow and outflow tracts of the left ventricle are shown the blood flows from the left auricle into the left ventricle through the inflow tract (posterior section of the left ventricular cavity, from mitral valve to apex) and then through the outflow tract (anterior section from apex to aortic valve) into the aorta

Summary—A case of inactive mitral and aortic stenosis is presented Longitudinal section of the heart demonstrates mitral stenosis with dilated left auricle and hypertrophied right ventricle aortic stenosis with hyper trophied left ventricle and also inflow and outflow tracts of the left ventricle

PLATE 15 INACTIVE MITRAL AND AORTIC STENOSIS



Left axis deviation

Anterior view Compare with roentgenogram Plate 1



Longitudinal section of the clockwi e rotated heart as in left oblique position

PLATE 15 INACTIVE MITRAL AND AORTIC STENOSIS

D S, male, white, dentist, aged 73

Admitted on June 6, 1944, for prostatectomy, performed four days later Died on June 19, 1944, of aspiration pneumonia

The heart weighed with aortic arch, pulmonary artery and its branches hila of the lungs, esophagus, and greater part of the thoracic aorta 1 050 grams It is estimated that the heart itself without these other structures *probably weighed 600 grams or more*

Longitudinal section of the heart rotated clockwise and placed in left oblique position the same as in left oblique view on the roentgenogram (see Plate 2) Pathological changes in various chambers of the heart are all shown in this one section mitral stenosis with dilated left auricle and hypertrophied right ventricle (4 to 10 mm normally, 3 to 4 mm) also aortic stenosis with *hypertrophied left ventricle (20 mm normally 12 to 14 mm) Both inflow* and outflow tracts of the left ventricle are shown the blood flows from the left auricle into the left ventricle through the inflow tract (posterior section of the left ventricular cavity from mitral valve to apex) and then through the outflow tract (anterior section from apex to aortic valve) into the aorta

Summary—A case of inactive mitral and aortic stenosis is presented *Longitudinal section of the heart demonstrates* mitral stenosis with dilated left auricle and hypertrophied right ventricle, aortic stenosis with hyper trophied left ventricle and also inflow and outflow tracts of the left ventricle

PLATE 16 MITRAL, TRICUSPID, AND AORTIC STENOSIS WITH CARDIAC FAILURE

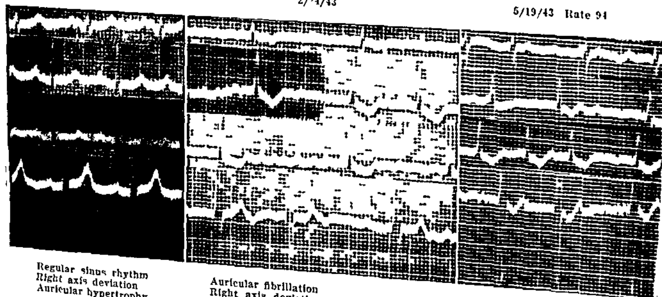
1/19/43 Heart rotated counterclockwise as in mitral stenosis

5/26/43 Rotated heart markedly enlarged as in cardiac failure Increased *shadow density* in the right base due to pulmonary infarct and hydrothorax

1/1 /43 Aged 3

2/24/43

5/19/43 Rate 94



Regular sinus rhythm
Right axis deviation
Auricular hypertrophy

Auricular fibrillation
Right axis deviation $ST_{2,3}$ depressed (digitalis?) $T_{2,3}$ inverted suggesting right ventricular strain

3 MIXED VALVULITIS

PLATE 16 MITRAL, TRICUSPID, AND AORTIC STENOSIS WITH CARDIAC FAILURE

L T, female, white, aged 37 Admitted on March 2, 1943, died on June 1, 1943

Patient had scarlet fever at age of 7, but never rheumatic fever "Heart trouble" at the time of second pregnancy in 1933 She had felt perfectly well until about five months before her present admission her complaints were of gradual onset Complaints on admission were dyspnea, palpitation, chest pain, productive cough, ankle edema, anorexia, and indigestion Heart enlarged, the left border in the anterior axillary line, auricular fibrillation, loud systolic and diastolic murmurs, most marked at the apex, heard over entire precordium Blood pressure 170/80 The liver was four finger breadths below the right costal margin Spleen not palpable

Her course in the hospital was one of progressive cardiac failure, marked by periods of disorientation and confusion, which began on the second hospital day She developed ascites and hydrothorax and these fluids were drained several times She had frequent episodes of fever and coughing, with frothy, blood tinged sputum from time to time—pulmonary infarcts

Roentgenograms —Heart markedly enlarged and rotated counterclockwise The normal left cardiac border consisting of aortic knob pulmonary artery and left ventricle disappeared posteriorly The upper portion of the left cardiac border is a straight aorta The middle portion which is usually concave is a straight line due to prominence and hypertrophy of the pulmonary conus of the right ventricle The lower portion is the anterior part of the right ventricle

A roentgenogram taken on May 26, 1943, shows the enlarged left auricle on the right cardiac border and the heart markedly enlarged as in cardiac failure

Electrocardiogram —Regular sinus rhythm right axis deviation auricular hypertrophy, replaced later by auricular fibrillation and right ventricular strain

PLATE 17 MITRAL, TRICUSPID, AND AORTIC STENOSIS WITH CARDIAC FAILURE (CONTINUED)



Mitral stenosis Hypertrophy and dilatation of the left auricle



Tricuspid stenosis Dilated and hypertrophied right auricle Dilated and hypertrophied right ventricle due to mitral stenosis



Aortic stenosis Left ventricular hypertrophy the wall is 1[illegible] mm in thickness Note a calcified nodule about [illegible] cm below the aortic valve

PLATE 17 MITRAL, TRICUSPID, AND AORTIC STENOSIS WITH CARDIAC FAILURE (CONTINUED)

L I female, white, aged 37

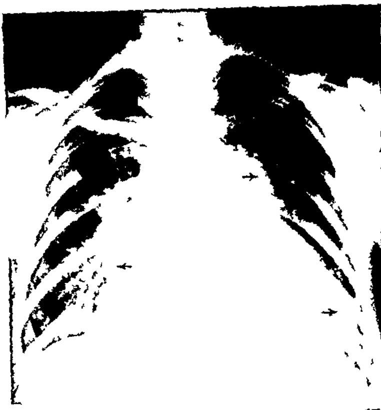
Autopsy—The chief change in the heart is valvular most marked at the mitral valve Chronic indurative mitral aortic, and tricuspid endocarditis *with extensive calcification and stenosis of aortic and mitral valves* Pulmonary valve is unaltered Hypertrophy and dilatation of left and right ventricles and auricles Chronic passive hyperemia of the lungs (brown induration) *liver (nutmeg), spleen and other abdominal viscera bilateral hydrothorax* (right 1 500 cc left 100 cc), ascites (5 000 cc) Thrombosis of pelvic veins, mural thrombi of both auricular appendages multiple pulmonary emboli with *multiple acute and organizing infarcts of the lungs and infarcts (early organizing)* of the spleen and kidneys The multiple pulmonary infarcts could have arisen from the right auricular thrombus and those in the spleen and kidneys *from the left auricular thrombus*

Sparse disseminated subacute periarteriolitis of the myocardium Disseminated subacute focal inflammation of the liver (portal spaces)

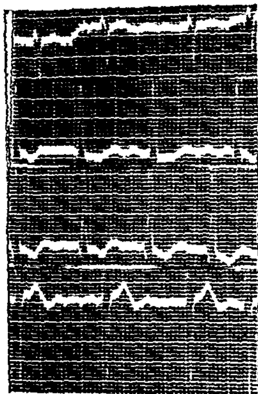
The interpretation of the post mortem findings are consistent with the primary disease of chronic rheumatic valvular heart disease with evidence of *left and right ventricular failures as evidenced by the chronic passive hyper*emia of the lungs on the one hand and of the liver and other viscera the hydrothorax and ascites on the other The only evidence of disseminated inflammation in the body suggestive of a rheumatic periarteritis is found in the myocardium and liver but because of the absence of significant inflammation elsewhere the significance of the periarteriolitis of the myocardium without characteristic Aschoff nodules is not interpreted as conclusive evidence of recurrent rheumatic inflammation

Summary — A case of rheumatic heart disease with involvement of mitral *tricuspid and aortic valves with predominance of mitral stenosis* and advanced heart failure is presented

PLATE 18 VALVULITIS OF ALL THE VALVES, CARDIAC FAILURE



3 20 43 Unusually prominent *pulmonary* conus suggests pulmonary stenosis



3/ 0/43 Aged 20 Rate 6 Auricular fibrillation Right ventricular strain Right axis deviation T_2 3 inverted



Mitral stenosis Dilated and hypertrophied left auricle



Aortic insufficiency Dilated and hypertrophied left ventricle

PLATE 18 VALVULITIS OF ALL THE VALVES, CARDIAC FAILURE

W. S., male, white, aged 20. Admitted on March 19, 1943; died on April 20, 1943.

Rheumatic fever at the age of 4; hospitalized for one year. He was well until the age of 10 when he had another severe attack of polyarthritis and precordial pains. A third attack of arthritis occurred at the age of 15 and lasted six months. He was well until the present hospital admission. Although he had a tonsillectomy at the age of 4, he had frequent sore throat; his right ear had drained for the previous seven years.

On admission patient complained of dyspnea of two months' duration, edema of legs for one month. Physical findings revealed orthopnea, cyanosis, normal temperature, bilateral perforated eardrums without discharge, carious teeth. Heart enlarged to right and left; systolic and diastolic thrills and murmurs over entire precordium. Auricular fibrillation as in mitral stenosis. Blood pressure 175/45 as in aortic insufficiency. Edema of the legs, sacral edema, tense abdomen, enlarged pulsating liver, as in tricuspid stenosis.

The course in the hospital was one of progressive heart failure marked early by development of pleuritic pain, nausea and vomiting. Temperature for the previous two weeks between 100 and 102° F. In the last week he developed thrombosis of the left femoral vein.

Roentgenogram.—The heart is enlarged and rotated counterclockwise. The enlarged left auricle is seen on the right cardiac border as increased shadow density. Due to counterclockwise rotation of the heart the aortic knob dis*appeared posteriorly and the lower left cardiac border is formed by the* anterior portion of the hypertrophied right ventricle. The unusually prominent pulmonary conus on the mid portion of the left cardiac border suggests marked hypertrophy of the conus as in pulmonary stenosis.

Electrocardiogram.—Auricular fibrillation. Right ventricular strain as in advanced mitral and pulmonary stenosis.

PLATE 19 VALVULITIS OF ALL THE VALVES, CARDIAC FAILURE (CONTINUED)



Pulmonary stenosis Right ventricle including pulmonary conus is markedly hypertrophied



Pulmonary stenosis Hypertrophy of the right ventricle



Tricuspid stenosis Dilated and hypertrophied right auricle



Fibrous adhesive pericarditis

PLATE 19 VALVULITIS OF ALL THE VALVES, CARDIAC FAILURE (CONTINUED)

W S, male, white, aged 20

Autopsy—Chronic, indurative mitral, aortic, tricuspid and pulmonary endocarditis with stenosis of mitral, pulmonic and tricuspid valves and insufficiency of the aortic valve Hypertrophy and dilatation of all cardiac chambers, especially of the left and right ventricles and the left auricle Fibrous adhesive pericarditis

Chronic passive hyperemia of the lungs liver, and abdominal viscera Recent thrombosis of the veins of the left lower extremity, extending to the bifurcation of the inferior vena cava Sparse pulmonary embolism—thrombi in each branch of pulmonary artery with small disseminated hemorrhagic pulmonary infarcts Moderate anasarca of the left lower extremity scrotum and sacrum, slight anasarca of the right foot Fatty changes of the liver cloudy swelling of the kidneys Slight icterus Bilateral disseminated fibrous pleuritis Sparse, acute, hemorrhagic bronchopneumonia

Summary — A case of rheumatic heart disease with involvement of all the valves including pulmonic, fibrous adhesive pericarditis and advanced heart failure is presented

The roentgenogram is characteristic of advanced mitral stenosis but the unusually prominent pulmonary conus on the left cardiac border is suggestive also of pulmonary stenosis in addition to mitral stenosis

The electrocardiograms with auricular fibrillation as in mitral stenosis and right ventricular strain correlate well with roentgenographic and clinical findings of advanced right heart failure

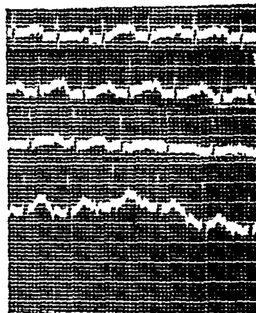
The autopsy showed the valves most involved were mitral and pulmonic (stenosis), with aortic insufficiency (blood pressure 175/45) and slight tricuspid stenosis next and also characteristic changes in all the chambers of the heart

PLATE 20 ACTIVE CARDITIS, WITH PREDOMINANT MYOCARDITIS, AND CARDIAC FAILURE



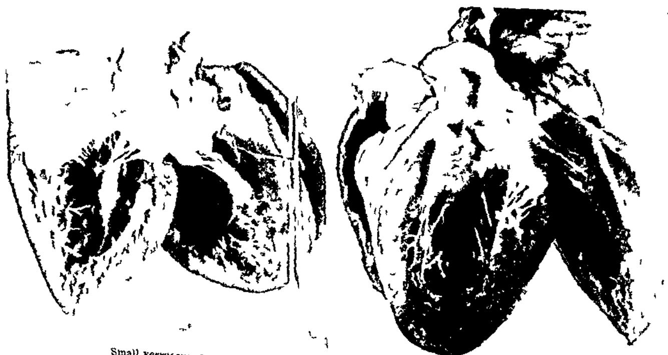
Heart enlarged in all directions

9/11/41 Aged 6



Rate 1?0 PR 0 ?0 second
Prolonged PR interval Incomplete AV block

All T's are low and with sinus tachycardia suggest myocardial insufficiency



Small verrucous vegetations on the mitral valve No significant aortic changes Dilatation of the left ventricle due to myocarditis Minimal fibrinous pericarditis

4 MYOCARDITIS

PLATE 20 ACTIVE CARDITIS, WITH PREDOMINANT MYOCARDITIS, AND CARDIAC FAILURE

P V, *female, white, aged 6 Admitted on July 30, 1941, died on Oct 22, 1941*

Swollen and painful wrists and ankles, five months before admission Pain and swelling in the joints of the hands and abdominal pain with occasional vomiting of one *month's duration Physical examination on admission re vealed* a poorly nourished child Pulse 116 respirations 20 blood pressure, 105/70 Decayed teeth, enlarged and reddened tonsils cervical adenopathy Heart enlarged to right and left palpable systolic thrill loud blowing systolic apical murmur, transmitted to axilla (mitral insufficiency) Liver enlarged one fingerbreadth below the costal margin Swollen fingers and ankles Sedimentation rate 34 millimeters

The course in the hospital was one of gradually progressive heart failure with enlargement of the heart and liver with edema A month after admis*sion the heart was enlarged to the anterior axillary line and a loud squeak*ing pericardial friction rub was heard at the apex

Roentgenograms taken on various dates showed moderate *diffuse cardiac* enlargement suggesting pericarditis or dilatation due to myocarditis

With a weak flabby myocardium the **electrocardiogram** showed prolonged PR interval and low voltage of all T s

Autopsy—Subacute rheumatic mitral and tricuspid endocarditis Subacute rheumatic myocarditis with pale flabby pinkish gray myocardium of *both ventricles and dilatation of the cardiac chambers (but without Aschoff* bodies microscopically) Minimal fibrinous pericarditis Petechial hemorrhages of the epicardium pleura and lining of the stomach Hydrothorax ascites anasarca Chronic passive hyperemia of the lungs liver spleen and kidneys Marked fatty changes of the liver

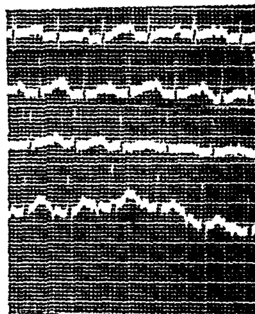
Summary—A case of active carditis with predominant myocarditis and cardiac failure is presented

PLATE 20 ACTIVE CARDITIS, WITH PREDOMINANT MYOCARDITIS, AND CARDIAC FAILURE



Heart enlarged in all directions

9/11/41 Aged 6



Rate 130 PR 0 ?0 second

Prolonged PR interval incomplete A V block

All T's are low and with sinus tachycardia suggest myocardial insufficiency



Small verrucous vegetations on the mitral valve No significant aortic changes Dilatation of the left ventricle due to myocarditis Minimal fibrinous pericarditis

PLATE 21 ELECTROCARDIOGRAM IN MYOCARDITIS (COMPOSITE ILLUSTRATION)

Myocardial damage is an anatomical and not a cardiographic diagnosis The electrocardiogram shows only changes in conduction, which, considering the clinical findings, may lead us to suspect myocardial damage

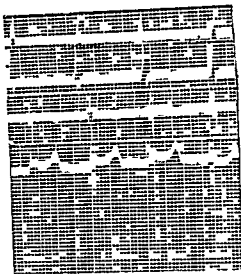
The walls of the auricles (changes in P), ventricles (changes in QRS, ST, T) or the *septum* (*auriculoventricular block, incomplete or complete, or bundle branch block*), may be involved One example of auricular myocardial involvement is that present in mitral stenosis with auriculitis and changes in P which later may lead to auricular fibrillation (see Plate 11) In contradistinction to myocardial infarction, where the myocardial damage is usually localized the rheumatic myocarditis is usually generalized of a disseminated type causing changes in all the leads with disturbance in intraventricular conduction as shown by wide slurred or splintered QRS, deformed ST, or low, inverted T's An associated myocardial insufficiency may cause low voltage of all leads and tachycardia If the septum is involved, incomplete block with prolonged PR and dropped beats complete block or bundle branch block may be present

Serial electrocardiograms may reveal changes indicating activity of myocardial damage Usually rheumatic myocarditis heals without any impairment of myocardial conduction and a normal electrocardiogram is found as *indication of complete healing In some cases the scar tissue may permanently* interfere with normal conduction Inverted T's or various forms of heart block remain permanent and unchanged for years as residue of an old healed damage

Electrocardiogram C—D S female white with a negative history on May 30 1924 aged 18 contracted upper respiratory infection with severe sore throat Temperature 102 F and pulse 100 followed in a few days by attacks of Adams Stokes syndrome with unconsciousness convulsions and heart rate as low as 15 Acutely ill for a few weeks she recovered and has been well since The complete auriculoventricular block found in June 1924 remained unchanged for over twenty two years and was still present in March 1947

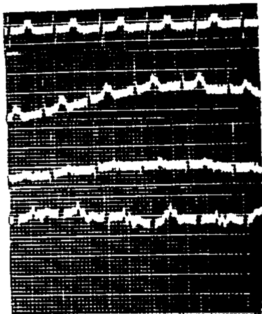
PLATE 21 ELECTROCARDIOGRAM IN MYOCARDITIS (COMPOSITE ILLUSTRATION)

A 5/15 4? Aged 4?



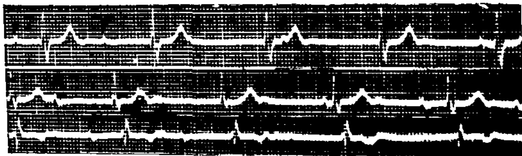
T1 is low voltage
T2 is inverted
QRS inverted

B 9/?/41 Aged 6



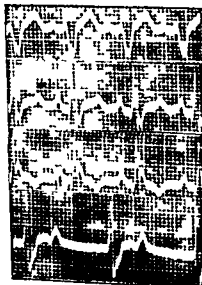
Rate 13? PR 0 ?4 second Low grade of incomplete block

C 3/6 3 Aged 31



Rate auricular 65 ventricular 46 No relationship between auricular (P) and ventricular (QRS T) contractions Complete heart block Present since June 19 4 to date (March 194?)

D 10/19/4 Aged 16



In the first electrocardiogram *(A)* rheumatic myocarditis affects the myocardium in general without affecting the bundle of His

The last three electrocardiograms *(B C D)* are examples of rheumatic myocarditis affecting the His bundle (special tissue in myocardium) causing either incomplete or complete auriculoventricular block or bundle branch block

5 PERICARDITIS

PLATE 22 PERICARDITIS WITH EFFUSION

A B, male, white, physician, aged 36

Upper respiratory infection and tonsillitis, followed about three weeks later by fever up to 101° F, *spasmodic cough without* expectoration, and dull lower substernal pain

Heart Apex beat neither visible nor palpable *heart enlarged to left and* right, heart sounds are audible, no murmurs A loud pericardial rub heard in lower sternal region Blood pressure, 118/78 pulse 140 regular Temperature, *99 6°* F Patient hospitalized for three weeks Pericardial rub disappeared at end of the first week Patient made an uneventful recovery

Roentgenograms — Anterior, left *oblique and right oblique views (see Plate* 23), demonstrate the absorption of fluid

Electrocardiograms —Electrocardiograms taken in this case showed a low voltage of QRS, T, and tachycardia (120 to 140), but normal a month later and ever since

The electrocardiographic abnormalities as low voltage or inversion of $T_{1, 2, 3}$ are valuable clues in doubtful or unsuspected cases of pericarditis The electrocardiographic changes are probably due to an associated myocarditis but may be due to compression of coronary vessels by pericardial fluid or adhesions and impairment of blood supply to the myocardium In the majority of cases when recovery takes place T's will be found normal again in others inversion of $T_{1, 3}$ may remain permanently as a sign of a scar tissue in the myocardium or pericardium which interferes with a normal myocardial conduction or normal *blood* supply *to the myocardium and as such is of no clinical* value Inversion of $T_{1, 2, 3}$ in an adult may suggest a possibility of coronary disease The correlation with clinical findings and history will clarify this point

Pathology —Plates 14 19 and 20 illustrate the gross pathology of the *fibrinous adhesive pericarditis as a residue of rheumatic carditis*

PLATE 22 PERICARDITIS WITH EFFUSION



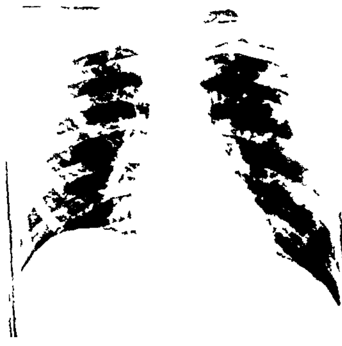
4/ ? 44 Cardiac shadow enlarged in all directions Loud pericardial rub heard in the lower sternal region



4/ 6/44 Cardiac shadow decreased in size Left base pleuritis Slight pericardial rub still present



5/5 44 Cardiac shadow near normal Pericardial rub gone L ft base pleuritis present



5/24/44 Cardiac shadow normal Left ba e pleuritis disappeared

PLATE 23 PERICARDITIS WITH EFFUSION (CONTINUED)

A. B., male, white physician, aged 36.

Roentgenograms taken in left and right oblique views illustrate the extent of pericardial fluid to the left and right posteriorly.

Left Oblique View.—The cardiac shadow is diffusely enlarged and markedly overlaps the spine as in marked left ventricular hypertrophy and dilatation (see Plate 31). Correlation with clinical findings will clarify the differential diagnosis.

Right Oblique View.—The cardiac shadow is diffusely enlarged and obliterates the entire retrocardiac space especially in the lower portion. In mitral stenosis (see Plates 9 and 12) the retrocardiac space is only partially compressed in the upper portion by the enlarged left auricle.

Summary.—A case of pericarditis with effusion is presented to demonstrate characteristic roentgenographic findings.

PLATE 23 PERICARDITIS WITH EFFUSION (CONTINUED)



4/ 44 Left oblique view Cardiac shadow enlarged post tiorly to left overlapping the pine



/44 Left oblique view Cardiac shadow normal



4 44 Right oblique view Cardiac shadow en larged pc teriorly to right obliterating retrocardiac pace



5/? /44 Right oblique view Cardiac shadow normal Retrocardiac space is clear

6 ACTIVE CARDITIS

PLATE 24 ACTIVE CARDITIS WITH CARDIC FAILURE AND PNEUMONIA

J R, male, white, aged 16 Admitted on Jan 25, 1944, died on Jan 29, 1944

Apparently well until five months before admission, when he had rheumatic fever with involvement of the heart, from which he apparently recovered He was well until the previous week, when he contracted pneumonia On admission, lungs full of rales and wheezes with decreased breath sounds in the right base, with heart sounds difficult to make out because of the noisy respirations and blowing murmurs obscuring the heart tones Blood pressure 130/70 Liver palpable almost to the navel abdomen distended Sputum disclosed type XIX pneumococcus Patient became progressively worse and died four days after admission

Autopsy —

1 Acute and chronic rheumatic carditis
   a Acute verrucous and chronic indurative aortic and mitral endocarditis with aortic stenosis and insufficiency mitral insufficiency and minimal indurative tricuspid endocarditis The acute changes are those of minute pinpoint to pinhead sized vegetations The chronic changes include adherence of the two leaflets at the commissures thickening and rolling back and shortening of the margins and leaflets with part of the leaflets held downward by thick tendinous cords
   b Acute subacute, and chronic rheumatic myocarditis, with Aschoff nodules
   c Acute and organizing serosanguinous and fibrinous pericarditis
   d *Hypertrophy and dilatation of the cardiac chambers*
2 Mild bilateral hydrothorax ascites, subacute passive congestion of the abdominal organs mild acute hyperplasia of the spleen
3 Acute pneumococcal lobular pneumonia involving all lobes and mild right fibrinous pleuritis

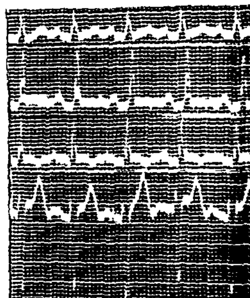
Summary —A case of active carditis with cardiac failure complicated by pneumonia is presented

PLATE 24 ACTIVE CARDITIS WITH CARDIAC FAILURE AND PNEUMONIA



Heart shadow enlarged in all directions Bilateral pneumonia

1/26/44 Aged 16 Rate 110



All QRS slurred QRS_4 inverted T low voltage T_4 inverted

Acute verrucous and chronic indurative *mitral* endocarditis

7 BACTERIAL ENDOCARDITIS

PLATE 25 BACTERIAL ENDOCARDITIS

It is customary on the basis of duration of the infection to distinguish two forms (1) acute as parts of sepsis, resulting from some local infection signs of sepsis usually outweigh those of cardiac involvements (2) subacute superimposed upon an old valvular deformity usually rheumatic or congenital more rarely arteriosclerotic or syphilitic

With the advance of penicillin the time element distinguishing the two forms has been markedly changed Also a number of cases of so called subacute bacterial endocarditis have been of very short duration due to fatal complications as for instance cerebral hemorrhage embolus or ruptured mycotic aneurysm Therefore it would be logical if clinicians would discard the words acute or subacute in diagnosing bacterial endocarditis

In addition to septic fever enlarged spleen positive blood culture and cardiac murmur the bacterial endocarditis manifests itself by various vascular phenomena (1) Emboli, as cerebral or Osler's nodes on fingers and toes (2) Arteritis with thrombosis and mycotic aneurysm as cerebral or coronary blood vessels (3) Hemorrhages as cerebral renal or petechiae If the above manifestations affect the vital organs they may cause death in spite of successful treatment of the bacterial endocarditis and sterilization of the blood stream with penicillin The same is true when bacterial endocarditis is complicated by cardiac failure

PLATE 25 BACTERIAL ENDOCARDITIS

SUBACUTE BACTERIAL ENDOCARDITIS

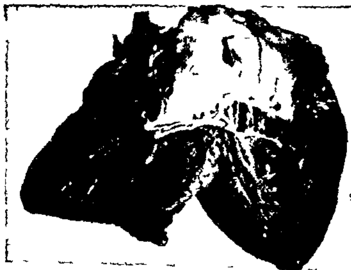
G S male white aged 5? Rheumatic fever at age 1?

Chronic indurative mitral and aortic endocarditis Acute thromboulcerative pneumococcal mitral and aortic endocarditis

The mitral leaflets show chronic changes and in addition acute changes consisting of a few small brown and pale rough vegetations Note small dark patches of subacute bacterial endocarditis disseminated above the margin of the mitral leaflets

The myocardium of the left ventricle is 0 to ?5 mm thick Petechial pericardial hemorrhages Thrombosis of the right auricular appendage

Death due to pulmonary infarct and aspiration bronchopneumonia





ACUTE BACTERIAL ENDOCARDITIS

C P male white aged 66

Cystitis with septicemia Large infected purulent vegetation on the middle aortic cusp Abscess of the left lung arachnoiditis with meningitis septic sple n

PLATE 26 BACTERIAL ENDOCARDITIS WITH CEREBRAL HEMORRHAGE

G B female white aged 32 Admitted on July 6, 1943 died on July 29 1943

Old rheumatic aortic insufficiency and mitral stenosis with loud double aortic and mitral murmurs water hammer pulse, blood pressure 140/10 Following tooth extraction, the patient developed bacterial endocarditis with petechiae and positive blood cultures for **Streptococcus viridans** *Short duration* of sickness due to fatal cerebral hemorrhage

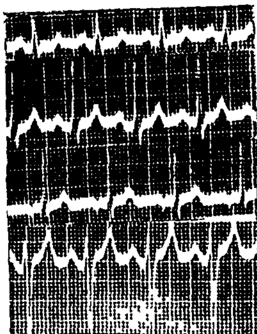
Autopsy —Chronic rheumatic mitral and aortic endocarditis with stenosis and insufficiency Streptococcal viridans mitral endocarditis Extensive sub arachnoid basal hemorrhage Numerous disseminated hemorrhages of the serosa of the stomach and intestines submucosal hemorrhages of the kidneys hemorrhages of the lungs Acute hyperplasia of the spleen

Note that rheumatic valvulitis affected mitral and aortic valves while bacterial endocarditis affected the mitral valve only

Summary — A case of bacterial endocarditis with positive blood cultures for **Streptococcus viridans**, superimposed upon an old rheumatic valvulitis and of short duration due to fatal cerebral hemorrhage is presented

PLATE 26 BACTERIAL ENDOCARDITIS WITH CEREBRAL HEMORRHAGE





Normal

Left midcardiac contour is straight—mitral stenosis Left lower cardiac border is out and rounded





Mitral valve

Aortic valve

Chronic rheumatic mitral and aortic endocarditis with stenosis and insufficiency
Acute Streptococcus viridans *mitral endocarditis* Subarachnoid basal hemorrhage

PLATE 27 BACTERIAL ENDOCARDITIS WITH MYCOTIC ANEURYSM

A P, male aged 38 Admitted on July 9 1940, died on August 16, 1941, after thirteen months of hospitalization

Old rheumatic mitral and aortic endocarditis Blood pressure, 116/60 Bacterial endocarditis with enlarged spleen, numerous petechiae nodules (emboli) on toes and fingers but always negative blood cultures during the thirteen months in the hospital Two months before death, patient developed acute serofibrinous pericarditis

Autopsy—Indurative mitral and aortic endocarditis Subacute bacterial mitral endocarditis Mycotic aneurysm and thrombosis of the circumflex branch of the left coronary artery Organizing obliterative pericarditis The maximum thickness of the wall of the left ventricle at the base is 14 *millimeters whereas the apex thins down to 4 millimeters* Extensive disseminated subpleural hemorrhages disseminated petechial hemorrhages of the ileum marked hyperplasia of the spleen old healed infarctions of the right kidney

Mycotic embolic aneurysm of a coronary artery results from softening of the arterial wall at the site of lodgment of an embolus secondary to bacterial endocarditis The proximal part of the left coronary is most frequently involved and rupture occurs in about one half of the cases It is usually single one to many centimeters in size (3 cm here) and the lumen may show partial or complete occlusion by a thrombus in various stages of organization

This case reminds us of one autopsied two years ago Male white aged 38 Subacute bacterial endocarditis superimposed upon a chronic indurative mitral endocarditis ruptured mycotic aneurysm of the left posterior cerebral artery *with extensive cerebral hemorrhage*

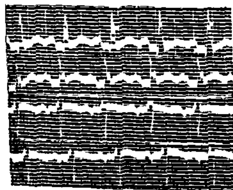
Summary — A case of bacterial endocarditis with mycotic aneurysm superimposed upon an old rheumatic valvulitis is presented Negative blood *cultures during thirteen months of hospitalization*

PLATE 27 BACTERIAL ENDOCARDITIS WITH MYCOTIC ANEURYSM

4/10/41 Left ventricular border is out to the left

7/11/41 Heart markedly enlarged in all directions Pericarditis

6 0/41 Aged 36



Left axis deviation QRS notched QRS_4 inverted T_4 isoelectric possibly a technical error as Lead 4 is similar to Lead 3



Indurative mitral and aortic endocarditis Subacute bacterial mitral endocarditis Organizing pericarditis Mycotic aneurysm with thrombosis of the left coronary artery

PLATE 28 BACTERIAL ENDOCARDITIS WITH SPONTANEOUS PERFORATION OF RIGHT AORTIC CUSP

T C male, Negro, aged 52 Admitted on Dec 26, 1937, died on March 19 1938

Admitted with complete urinary retention, secondary to benign prostatic hypertrophy unrelieved for two days because of unsuccessful attempts at self catheterization Temperature 100 2 F , blood pressure 190/100 Heart normal no murmurs On the eighth day suprapubic cystotomy was performed The postoperative course can be divided into four periods

1 The first period of sixteen days from the day of cystotomy to the day *when* **Bacillus coli** *appeared on blood culture* Septic temperature

2 The second period of twenty two days from the day of positive blood culture to the day of appearance of a loud systolic apical murmur

3 The third period of twenty two days from the day of appearance of heart murmur to the day of cerebral embolism (spastic extremities) Petechiae of the conjunctivae and skin were noted

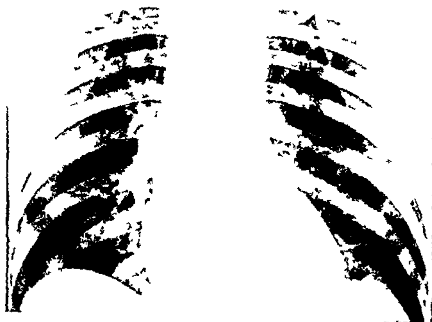
4 The fourth period of eight days from the day of cerebral embolism to the day of death

Autopsy —Slight hyperplasia of the prostate gland fibrous periprostatitis unhealed cystotomy wound acute cystoureteritis

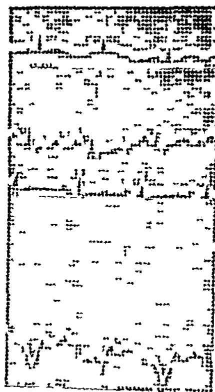
Acute thromboulcerative aortic and mitral endocarditis spontaneous perforation of right aortic cusp Septic infarcts of the spleen left kidney cerebrum and myocardium Petechial hemorrhages of the skin and small intestines Slight bilateral hydrothorax ascites and anasarca Slight generalized arteriosclerosis Hypertrophy of the left ventricle

Summary —A case of bacterial **(Bacillus coli)** endocarditis following acute genitourinary infection is presented Note septic temperature positive blood cultures appearance of heart murmurs cerebral embolism and infarct petechial hemorrhages of the skin and spontaneous perforation of right aortic cusp

PLATE 28 BACTERIAL ENDOCARDITIS WITH SPONTANEOUS PERFORATION OF RIGHT AORTIC CUSP



Normal



T_1 $_2$ $_3$ low T_4 inverted (normal with old technique of taking Lead 4)



Bacterial mitral and aortic endocarditis following acute genitourinary infection Vegetations and thrombi on aortic cusps perforation of right aortic cusp slight chronic adherence of posterior and left cusps Slight perforation of anterior mitral leaflet a vegetation on its auricular surface

III

ARTERIOSCLEROTIC HEART DISEASE

PLATE 29 ATHEROSCLEROSIS OF THE AORTA

The atherosclerotic process involves the aorta, mitral and aortic valves, coronary arteries, and may be associated with abnormal rhythm in the auricles (auricular flutter, or fibrillation).

In the atherosclerosis of the aorta the intima is altered by atheromatous plaques and calcification with increased severity from the arch down toward the abdominal aorta. The weakened wall may dilate, causing a diffuse dilatation and tortuosity of the aorta, more frequently so when associated with hypertension. Clinically this condition is unnoticed by the patient and does not cause any symptoms unless rupture of the weakened aortic wall occurs. It may rupture completely and cause a fatal hemorrhage or may rupture partially with dissection of the coats of the vessel by blood. In dissecting aneurysm death may not occur immediately but be postponed for days, weeks or even months. To complete the discussion of aneurysms we included a short description of arteriovenous and cirsoid aneurysms (*Plate 33*) which are not due to arteriosclerosis.

The atherosclerosis of the mitral valve may cause mitral insufficiency with a soft systolic apical murmur, usually of little consequence to the patient.

The atherosclerosis of the aortic valve may cause aortic insufficiency with a soft diastolic aortic murmur and characteristic blood pressure changes. The condition and result are in all respects similar to aortic insufficiency of rheumatic etiology and for further details we refer the reader to that section. Fibrosis and sclerosis may cause slight aortic stenosis with soft systolic aortic murmur or nodular calcifying aortic stenosis with pronounced blowing systolic aortic murmur, small plateaulike pulse, normal or slightly lower blood pressure and marked hypertrophy of the left ventricle.

Atherosclerosis of the coronary arteries will be discussed in detail later.

PLATE 29 ATHEROSCLEROSIS OF THE AORTA

L D female white aged 0

The lining of the aorta is *extensively altered by gray* and yellow atheromatous plaques with considerable calcification and with increa ed *severity from arch down* ward until at the bifurcation there is a partial occlusion by *yellow* and predominantly red tan mural thrombus In addition there are numerous small thin reddish brown mural thrombi forming conspicuous soft nodules upon the plaques in thoracic and *abdominal* aorta





W A *male* white aged 65

Ruptured aneurysm of abdominal *aorta* Extensive generalized arteriosclerosis in cluding *the* aorta pulmonary and *coronary* arteries Benign nephrosclerosis

PLATE 30 ATHEROSCLEROSIS OF THE AORTA, RUPTURED ABDOMINAL AORTIC ANEURYSM

B C, male, white, aged 85 Admitted on Oct 6, 1941, died on Dec 7, 1941

Patient had an aneurysm of abdominal aorta for the preceding three years, according to his physician's statement Four hours before admission patient had a sharp pain in the left lower quadrant slight nausea, desire to defecate, pallor and sweating Two hours later, he felt slightly better Blood pressure, 118/102 pulse of fair quality auricular fibrillation On admission patient appeared acutely ill with pallor of the face and cold extremities Pulse 104 temperature 98 6° F respirations 36 blood pressure, 82/48 A pulsating mass was palpated in the abdomen extending downward to the bifurcation of the aorta, with increased tenderness in the left lower abdomen Three hours after admission patient appeared to be in extreme shock the entire abdomen felt very full as though with fluid and patient died soon after

Autopsy —Atherosclerosis of the aorta aneurysm of the lower part of abdominal aorta with mural thrombosis Spontaneous perforation of the aneurysm with retroperitoneal hematoma and hemoperitoneum Atherosclerosis of the coronary arteries slight fibrosis of myocardium minute scars and organizing infarctions of the myocardium The thoracic portion of the aorta is tortuous Beginning at the superior end of the thoracic aorta and extending down along the posterior wall to the level of the diaphragm, at least 90 per cent of the lining is altered by soft yellowish brown elevations some of which are embedded in ulcerative regions

Summary —A case of atherosclerosis of the aorta with ruptured aneurysm of abdominal aorta in a man 85 years of age is presented

PLATE 30 ATHEROSCLEROSIS OF THE AORTA, RUPTURED ABDOMINAL AORTIC ANEURYSM



Dilated descending aorta seen as a diffuse shadow with a strai ht border extending from the aortic knob down and to the left of the pulmonary artery Within this shadow th irregular hilum may be seen

The interrupted short lines indicate the outline of the pulmonary artery



Senile dilatation of the heart Slight adherence of posterior and left aortic cusps Senile dilatation of ascending aorta Atherosclerosis of thoracic and abdominal aorta Aneurysm with mural thrombu i of lower part of abdominal aorta Spontaneous perforation of the aneurysm Hemoperitoneum

PLATE 31 SCLEROSIS OF THE ABDOMINAL AORTA

Roentgenograms of the spine and pelvis, anterior and lateral views, visualize the sclerosis of the abdominal aorta and iliac arteries.

Chest roentgenograms, anterior and left oblique views, visualize the aortic arch and the thoracic aorta.

In anterior view (Plate 1) the ascending aorta is seen on the right upper border, while the aortic knob is on the left. If aorta is dilated and tortuous, the ascending aorta on the right is bulging, the aortic knob *may be prominent with calcification noted on the border*; the descending aorta is a diffuse shadow with a straight border extending from the aortic knob down and to the left of the pulmonary artery. Frequently the tortuous course of the thoracic aorta may be followed within the cardiac shadow down to the diaphragm.

In left oblique view (Plate 2) the aortic arch is seen over the left auricle and bifurcated trachea. The dilatation of the aortic arch is plainly noticeable on the left oblique view as a wide round shadow over the heart and overlapping the spine (see Plate 31).

PLATE 31 SCLEROSIS OF THE ABDOMINAL AORTA



Anterior view Sclerosis of pelvic arteries and those of the thighs

Lateral view Marked sclerosis of the entire abdominal aorta

G S male white aged 61

Marked osteo-arthritic lipping in the bodies of the lumbar vertebrae Decalcification in the bones of the pelvis and lumbar vertebrae

PLATE 31 SCLEROSIS OF THE ABDOMINAL AORTA

Roentgenograms of the spine and pelvis, anterior and lateral views, visualize the sclerosis of the abdominal aorta and iliac arteries.

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PLATE 31 SCLEROSIS OF THE ABDOMINAL AORTA



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Lateral view Marked sclerosis of the entire abdominal aorta

G S male white aged 61

Marked osteo arthritic lipping in the bodies of the lumbar vertebrae Decalcification in the bones of the pelvis and lumbar vertebrae

PLATE 32 DISSECTING AORTIC ANEURYSM

R B male, white, aged 58 *Admitted on Dec 10, 1942, died on Dec 12 1942*

Patient had been in good health had never suffered from any cardiac ailment and as far as he knew had never had high blood pressure On the day before admission he was under emotional strain While reading a newspaper he was seized with a sudden pain in the cervical spine region it was not excruciating, but rapidly increased in intensity and spread down the thoracic spine as low as the scapula and then radiated bilaterally about the chest The pain was about the intensity of a toothache and seemed to throb He was unable to sleep that night and he had an urge to defecate, associated with pain and urinary frequency every three hours His pulse remained regular and firm there was no dyspnea palpitation, or cough

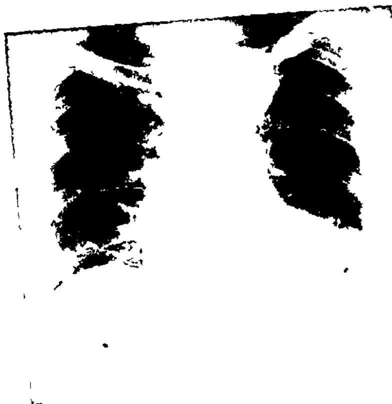
On physical examination, he did not appear acutely ill or in great distress Pulse 104, regular, heart and lungs negative blood pressure 200/124 There was no tenderness along the spine Dissecting aneurysm, diagnosed on the basis of clinical history Shortly after admission, patient complained of blurred vision later he vomited but no nausea or discomfort accompanied the vomiting On following day he felt much better Blood pressure 165/110 Death came suddenly, within five minutes of the onset of severe dyspnea

Autopsy—Chronic aortitis tear of the aorta dissecting aneurysm with perforation and massive left hemothorax displacement of mediastinum to the right compression atelectasis of the lungs

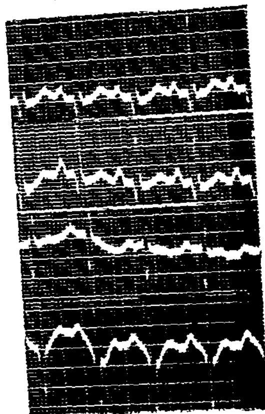
Summary—A case of dissecting aortic aneurysm is presented

PLATE 32 DISSECTING AORTIC ANEURYSM

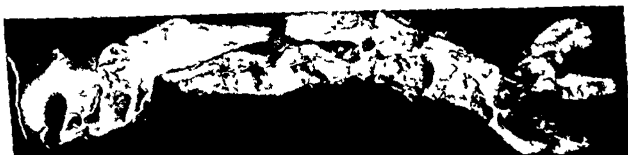
12/10/42 Aged 59 Rate 110



1 /10/42 Prominent aortic knob



ST1 2 3 deformed



Atherosclerosis of the aorta Dissecting aneurysm of the thoracic aorta with perforation and massive left hemothorax Linear transverse tear near the side of obliterated ductus arteriosus The adventitia is dissected away from the media down to the bifurcation of the abdominal aorta and into the right common iliac artery

PLATE 32 DISSECTING AORTIC ANEURYSM

R B male, white aged 58 Admitted on Dec 10 1942, died on Dec 12, 1942

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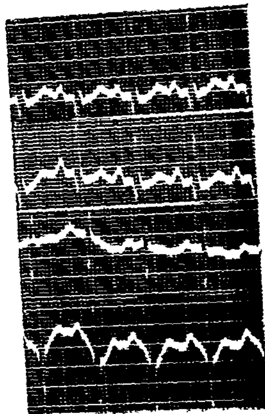
Summary—A case of dissecting aortic aneurysm is presented

PLATE 32 DISSECTING AORTIC ANEURYSM

12/10/42 Aged 58 Rate 110



1?/10 4? Prominent aortic knob



ST₁ ↓ deformed



Atherosclerosis of the aorta Dissecting aneurysm *of* the thoracic aorta with *perfora* tion and massive *left hemothorax* Linear transverse *tear* near the side of *obliterated* ductus arteriosus The adventitia is dissected away *from* the media down to the bifurcation of the abdominal aorta and into the right common iliac artery

PLATE 33 CIRSOID AND ARTERIOVENOUS ANEURYSMS

I C, male white aged 64 Admitted on March 22, 1942 died on May 11 1942

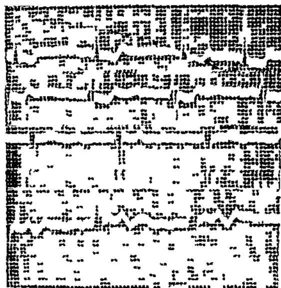
Advanced arteriosclerosis Blood pressure, 230/160

Illustrating a cirsoid (vinelike) aneurysm of the left common iliac artery altered by an acute red thrombosis of its lumen This aneurysm formed proximal to an arteriovenous communication between the left femoral artery and vein (arteriovenous aneurysm) due to a stab wound in the region fifty two years previously at age 12

The arteriovenous aneurysm manifested itself by a continuous murmur in the region of the aneurysm due to a continuous flow of blood from the artery into the vein

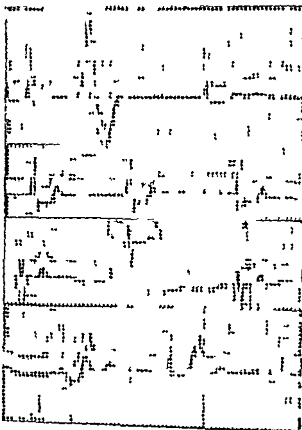
Ligation of the arteriovenous aneurysm was followed by circulatory disturbance in the left lower extremity complicated three weeks later by gas gangrene infection for which amputation of the extremity below the knee was done Death occurred five days after the amputation

1/26/40 Aged 62



Ra e 6 Auricular fibrillation Left axis deviation

3/23/42 Aged 64 B P 30/160



Rate 54 Auricular fibrillation Ventricular premature systole (Lead 1)



Thrombosed cir oid aneurysm of the left common iliac artery formed proximal to femoral arteriovenous aneurysm due to stab wound of 42 years duration

PLATE 34 NODULAR CALCIFYING AORTIC STENOSIS WITH CARDIAC FAILURE

C P, male, white, aged 68 Admitted on Oct 31, 1942, died on Dec 1942

Complaints on admission were dyspnea on exertion of three years du tion at rest for one year, ankle edema one month, tenderness in the ri upper quadrant and productive cough one week Blood pressure, 132/ Dullness and rales over the lower lobes The heart was greatly enlarg There was a loud aortic systolic murmur in the third interspace to the l of the sternum The liver extended 4½ inches below the right costal marg *There was fluid in the abdomen No history of rheumatic fever*

Patient was getting along fairly well, when two days before death developed pulmonary infarction Pulse, 130, temperature 102 2 F blo pressure, 140/100

Autopsy—Chronic nodular calcifying aortic endocarditis stenosis le ventricular hypertrophy *Chronic passive hyperemia of the lungs and a* dominal viscera ascites and right hydrothorax Thrombosis of the perivesic plexus of veins *multiple small pulmonary emboli with multiple small hem* orrhagic infarcts General arteriosclerosis chronic fibrous and calcifyin pleuritis

Heart—There is a marked irregular nodular rough calcification whic is chiefly on the aortic surface The stenosis is marked Calcified nodule *on the free margins and the cusps with thrombotic ulceration of one of th* nodules Agglutination of the commissures between the right and posteric cusps

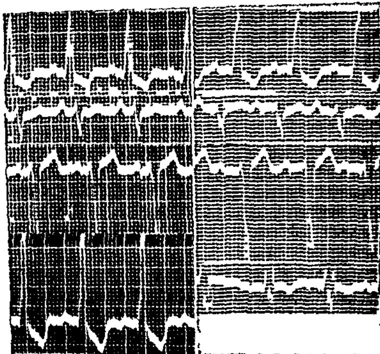
Summary—A case of nodular calcific aortic stenosis with cardiac failur is presented Marked hypertrophy of the left ventricle due to aortic stenosis and marked dilatation of the left ventricle with cardiac failure correlat well with electrocardiographic findings of left ventricular strain Death wa due to pulmonary emboli and infarcts

PLATE 34 NODULAR CALCIFYING AORTIC STENOSIS WITH CARDIAC FAILURE



11/3/4? Heart markedly increased in all directions Left ventricular border rounded and out Calcific plaque left pleura Increased lung markings

11/4/4? Aged 6?   11/ ?/4 BP 1 /??



Left axis deviation QRS relatively high ST₁ + d pre ed deformed fused with inverted T₁ Left ventricular strain
Similar to 11 4/42 except QRS₁ low notched ST₁ normal T₁ inverted low probab y due to coronary clerosi



Chronic nodular calcifying aortic stenosis Coronary sclerosis Dilatation and hypertrophy of the lef ventricl ?3 mm thick at the ba e



Enlarged view of the nodular calcific aortic stenosis

PLATE 35 ARTERIOSCLEROSIS, AURICULAR FLUTTER AND FIBRILLATION

The abnormal auricular, "circus" rhythm is due to abnormal initiation and transmission of auricular impulses

In **auricular flutter** the auricular activity is represented by flutter "F," waves large, regular, continuous deflections, which are uniform except where distorted by QRS, T occurring at the rate of from 200 to 380 per minute, usually recorded best in Leads 2 and 3 QRS occurs regularly at a rate which is slower than, and in a constant ratio to the auricular rate, as 2 to 1 3 to 1, or 4 to 1 In some cases especially when treated the ratio varies in the same tracing, and ventricular rhythm is irregular Rarely, the ratio may be 1 to 1 and such an electrocardiogram is distinguished with difficulty from those of other supraventricular tachycardias

In **auricular fibrillation** the auricular activity is represented by "f" waves small, irregular variable deflections which have usually a rate of 450 per minute QRS occurs at irregular intervals and as some ventricular contractions are weak and do not reach the radial pulse there will be a pulse *deficit*

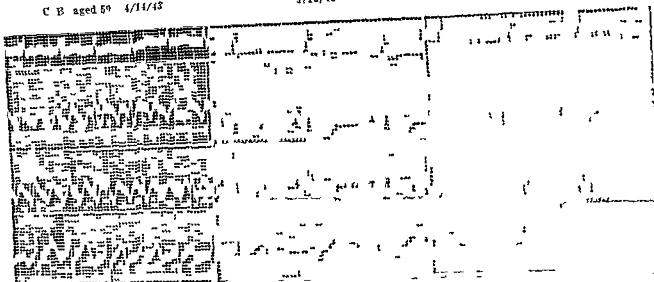
When auricular fibrillation is associated with a complete auriculoventricular heart block with ventricles responding to their own intraventricular center the ventricular rhythm is regular the heart rate is slow and there is no pulse deficit

The electrocardiogram shows "f" waves instead of P and regular QRS of intraventricular origin with slow ventricular rate If "f" waves are not visible, absence of P and regular QRS may suggest nodal rhythm and at times differentiation may be difficult especially when the ventricular rate is slow

The premature systoles which are frequently encountered in auricular fibrillation are of ventricular origin as the auricles while *fibrillating* are unable to initiate normal or premature (auricular or nodal) contractions

PLATE 35 ARTERIOSCLEROSIS, AURICULAR FLUTTER AND FIBRILLATION

C B aged 59 4/14/43 — 9/16/43 — 1 /15 43

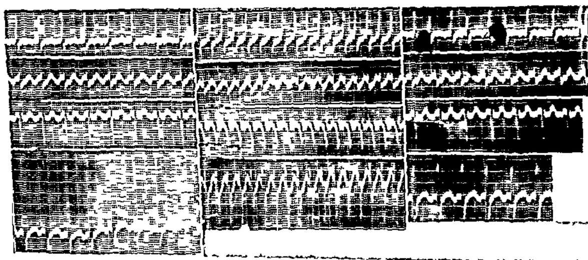


Auricular flutter of varying ratio (2 or 3 to 1) Ventricular rate 13

ST₂ s concave fused with T s digitalis effect Ventricular rate ?

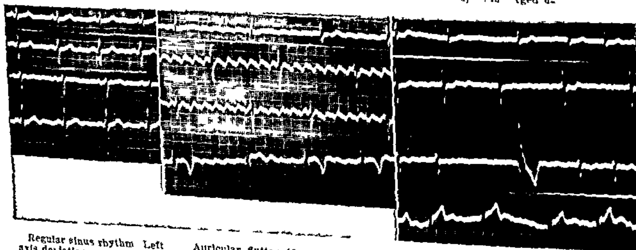
Normal tracing Died of pneu monia 4/14/44

F L aged 61



While first ECG (auricular flutter 2 1 rate of 125) was being taken patient had a paroxysmal attack of 1 1 *flutter with rate of* 250 which in a few minutes returned to 2 1 with rate of 125 Patient died five weeks later

W R aged 58 3 16/39 — 9/ 42 — 6/22/43 Aged 6-



Regular sinus rhythm Left axis deviation

Auricular flutter (6 or 5 1) QRS T₄ inverted Coronary disease

Auricular fibrillation Ventricular premature systole QRS₄ inverted T₄ up

PLATE 35 ARTERIOSCLEROSIS, AURICULAR FLUTTE AND FIBRILLATION

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In **auricular fibrillation** *the auricular activity is represented by* 'f' wa small, irregular, variable deflections which have usually a rate of 450 minute QRS occurs at irregular intervals and as some ventricular c tractions are weak and do not reach the radial pulse there will be a pu deficit

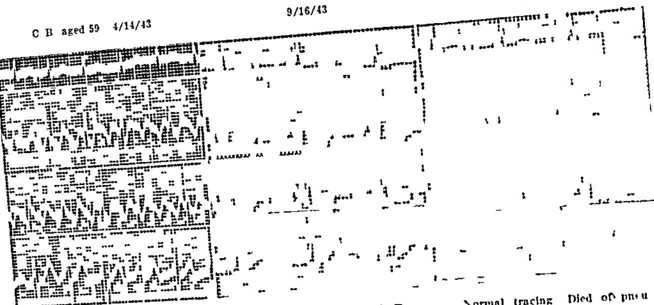
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PLATE 35 ARTERIOSCLEROSIS, AURICULAR FLUTTER AND FIBRILLATION

C B aged 59 4/14/43 9/16/43 1?/15/43

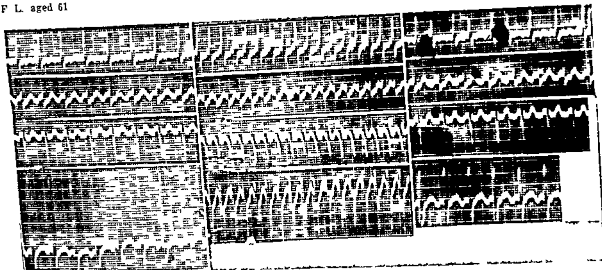


Auricular flutter of varying ratio (? or 3 to 1) Ventricular rate 1 ?

ST s concave fused with T a digitalis effect Ventricular rate

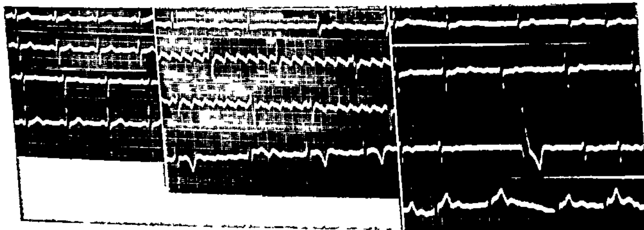
Normal tracing Died of pneu monia 4/14/44

F L. aged 61



While first ECG (auricular flutter ? 1 rate of 1?5) was being taken patient had a paroxysmal attack of 1 1 flutter with rate of 250 which in a few minutes returned to 1 with rate of 12 Patient died five weeks later

W P a ed 59 3 16/39 9/2?/4 6/ 43 Ag d 6



Regular sinus rhythm Left axis deviation

Auricular flutter (6 or 1) QRS, T inverted Coronary disease

Auricular fibrillation Ventricular premature systole QRS inverted T up

PLATE 35 ARTERIOSCLEROSIS, AURICULAR FLUTTER AND FIBRILLATION

The abnormal auricular "circus" rhythm is due to abnormal initiation and transmission of auricular impulses

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The premature systoles which are frequently encountered in auricular fibrillation are of ventricular origin as the auricles while fibrillating are unable to initiate normal or premature (auricular or nodal) contractions

disappear as soon as the redness and swelling in the area surrounding the myocardial infarct will subside

In myocardial infarct, especially when extensive, the balance of the left ventricular myocardium may fail, the myocardial insufficiency in this case being the result of a marked coronary insufficiency Clinically, there will be dyspnea as a sign of left ventricular failure The electrocardiogram will show tachycardia marked left axis deviation low voltage of all the leads (QRS T), or the ST s in Leads 1 and 4 or 2 and 3 instead of high take off or elevation may be markedly depressed, concave and fused with T's

In some cases, the myocardial infarct may be the focus for cardiac irritability and cause appearance of premature systoles or paroxysmal auricular tachycardia, flutter or fibrillation

The localized necrosis in myocardium leads to a localized involvement of endocardium with formation of a mural thrombus and of pericardium with pericarditis The thrombus may lead to emboli in systemic circulation and if a vital organ, as the brain is involved, *may be the* direct cause of death, while the myocardial infarction itself was clinically improving The localized pericarditis may cause a pericardial rub, heard usually for a short time anteriorly, in anterior infarct When the pericarditis is extensive not localized, the pericardial rub may be present over a long period of time, and also may be heard in posterior infarct

The necrotic myocardium *may* rupture If the wall ruptures, the hemorrhage may cause heart tamponade and death

The rupture of the interventricular septum may heal without any ill effect except for appearance *of a* systolic murmur in the region of the third or fourth left interspace near the sternal border While the congenital interventricular septum defect is in the weakest, membranous upper portion of the *septum*, the acquired septum defect is in the muscular portion In *congenital* septum defect, if associated with pulmonary stenosis and increased pressure in the right ventricle, the venous blood of the right ventricle flows into the left ventricle, mixes with arterial blood and causes central *cyanosis* In acquired septum

CORONARY SCLEROSIS AND MYOCARDIAL INFARCTION

Atherosclerosis of the intima of the coronary arteries is a process similar to that in any other arteries, but the changes resulting from it are of great importance as to the proper function of the myocardium

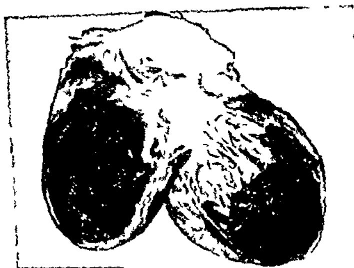
The coronary sclerosis may be silent and inactive with slight changes in QRS, ST, and T as the only evidence, if conduction in ventricular myocardium is impaired, or various types of heart block, incomplete, complete, or bundle branch block may be present, if the septum is involved

The coronary sclerosis may be active Clinically it will manifest itself by dull or sharp precordial pain on exertion or angina pectoris as due to insufficient blood supply and myocardial anoxemia, or dyspnea on exertion due to myocardial insufficiency and left ventricular failure In the electrocardiogram there will be noted changes in various abnormalities found before as inverted QRS, T's or ST changes when compared with previous electrocardiograms, those comparative changes are electrocardiographic signs of activity of coronary sclerosis

Atherosclerosis of the intima of the coronary arteries may lead to thrombosis and occlusion of the lumen of a branch of a coronary artery, and this in turn to a localized myocardial infarction and necrosis This is most frequently encountered in two locations (1) in the apex, anterior part of the left ventricle and adjoining portion of the interventricular septum, so called anterior infarct, a part supplied by anterior descending branch of the left coronary artery and, (2) in the base, posterior part of the left ventricle and adjoining posterior portion of the interventricular septum, so called posterior infarct a part supplied by the circumflex branch of the left coronary or by the right coronary artery

In early stage of infarction, the electrocardiogram shows marked changes in QRS, ST, and T, especially elevation and high take off of ST in Leads 1 and 4 in anterior, and Leads 2 and 3 in posterior infarcts Quite often, the myocardium in the vicinity of the necrotic area may also be involved, being red and swollen and increasing the interference with myocardial conduction Therefore, it is not unusual to find in the first day or two of infarction extensive electrocardiographic changes, including various degrees of heart block, which will

PLATE 36 RECENT AND OLD ANTERIOP MYOCARDIAL INFARCTS WITH LARGE MURAL THROMBUS



F M white male aged 6/

Acute coronary thrombosis a year ago advanced cardiac failure in the last three month Recent (silent) anterior infarct with a large organizing mural thrombus superimposed upon an (one year) old healed anterior infarct with ventricular aneurysm Marked dilatation of the left ventricle advanced cardiac failure chronic passive hyperemia of the lungs liver spleen and other viscera bilateral hydrothorax hydropericardium ascites and edema of right ankle Recent thrombosis of the periprostatic veins Large recent pulmonary infarct

defect, the same as in congenital without pulmonary stenosis the pressure in the left ventricle remains higher than in the right, the arterial blood from the left ventricle flows into the right, and there will be no central cyanosis

The active myocardial infarction may heal without leaving any clinical or electrocardiographic evidence of impairment In others, the infarcted area may cause localized thinning and bulging of the wall, a ventricular aneurysm, and impairment in myocardial conduction, with permanent changes in QRS, ST or T as residue of a healed infarct, and clinically dyspnea as a sign of left ventricular insufficiency and cardiac failure, which, if unrelieved, may gradually progress to advanced cardiac failure and death In advanced cardiac failure it is not unusual to find a thrombosis in some peripheral vein causing pulmonary embolism with infarction and death

In conclusion it is worth mentioning that one episode of myocardial infarction may be followed later by another in the same or another area, with the consequences already stated

PLATE 37 RECENT ANTERIOR INFARCT WITH PAROXYSMAL AURICULAR FIBRILLATION

1? [illegible] /39 1?/29/39 1?/30/39 ?/14/40



Rate 110
Left axis deviation
Q present ST high take off and fused with T

Rate 10
I aroxy mal auricular fibrillation

I ate 110
Regular sinus rhythm
I high peaked
Auricular hypertrophy

Rate 115
QRS low ST near normal T low inverted

QRS inverted ST markedly elevated and fu ed with T

Very slight ECG changes in QRS ST and T over period of six weeks



/2/40 Left ventricular hypertrophy Increased lui g markings—cardiac

Organizing myocardial infarct of the anterior wall of the left ventricle and septum with mural thrombus

PLATE 37 RECENT ANTERIOR INFARCT WITH PAROXYSMAL AURICULAR FIBRILLATION

W R, male, white, aged 57 Admitted on Dec 26, 1939, died on Feb 15, 1940

Hypertension known to be present since June, 1937 The chief complaint on admission was *constant, severe pain under the lower part of the sternum* of a few hours' duration, unaffected by bed rest and only slightly relieved by morphine The pain radiated toward the right shoulder and arm There was constant nausea and the patient vomited a greenish liquid a few times at home A severe dyspnea set in shortly after the pain began On admission, patient was breathing with difficulty, the general color was good the lips and hands were only moderately cyanotic Heart moderately enlarged to left, the rate was regular the sounds of good quality no murmurs Blood pressure 172/106 Leucocyte count 16 650 Temperature reached the peak of 102° F two days after admission and remained between 98 6 and 100° F for the rest of the course in the hospital The pulse varied from 100 to 130 respirations from 20 to 35

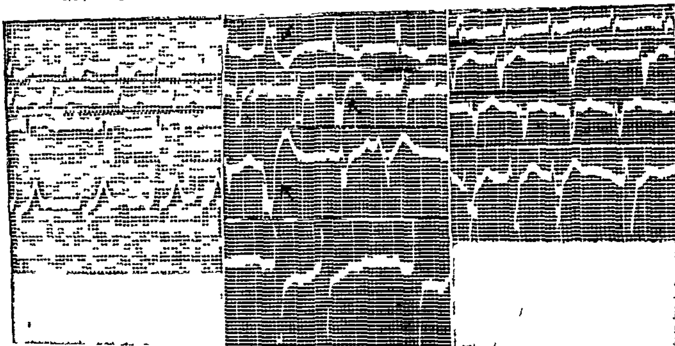
On Dec 28 1939, there was an arrhythmia and friction rub heard under the sternum Next day the pain disappeared there was pulse deficit a continued substernal friction rub was heard Nine days after admission the dyspnea increased in severity The condition was unchanged until Feb 14 1940 when the patient suddenly expired

Autopsy—Extensive organizing infarct of the anterior wall of the left ventricle and ventricular septum Extensive mural thrombus of the left ventricle Marked sclerosis and recanalized lumen of the descending branch *of the left coronary artery Generalized arteriosclerosis* Bilateral hydrothorax with slight compression atelectasis of the lower lobes Cyanotic induration of the lungs

Summary—A case of recent anterior myocardial infarct with typical electrocardiographic findings in Leads 1 and 4 is presented Paroxysmal auricular fibrillation appeared two days after admission and disappeared two days later There were very slight electrocardiographic changes noted over a period of six weeks and the patient died of left ventricular failure due to extensive myocardial infarction

PLATE 38 RECENT ANTERIOR MYOCARDIAL INFARCT WITH CARDIAC FAILURE

5/15/43 Aged 70 | 6/7/43 B P 170/100 | 6/15/43 Rate 11°



T diphasic

Ventricular premature systoles T_1 deeply inverted coved ST_4 depressed T isoelectric

QRS_1 low QRS_4 inverted T_1 I o electric T_4 low

Auricular fibrillation and left axis deviation present in all three tracings Changes in Leads 1 and 4 (QRS ST T) on various dates indicate recent anterior myocardial infarct Auricular fibrillation was present before the infarction



Left ventricle opened from the front showing aorta and aortic valve Mitral valve to the right Large recent organizing infarct apex of the left ventricle



Left ventricle opened from the back showing left auricle mitral valve Mural thrombus of left ventricle and left auricular appendage

The wall of the left ventricle in infarcted area is only 8 mm thick at the base 0 mm left ventricular hypertrophy due to associated *hypertension* (left axis deviation in electrocardiogram)

PLATE 38 RECENT ANTERIOR MYOCARDIAL INFARCT WITH CARDIAC FAILURE

J C, male, white, aged 70 Admitted on June 8, 1943, died on June 21, 1943

Patient had been in good health for about three months prior to admission when he began to have cramping precordial pain radiating down to left arm, on exertion or emotion lasting but a few minutes The pains were ascribed by his physician to coronary disease At 1 A M on the day of admission he was awakened by a severe pain, not relieved by morphine He was admitted early in the morning, however, without much pain On admission pulse, 100 temperature, 100 F blood pressure 170/100 Left cardiac border 2 cm behind the midclavicular line auricular fibrillation good cardiac sounds with a precordial systolic murmur Leucocyte count 20 000

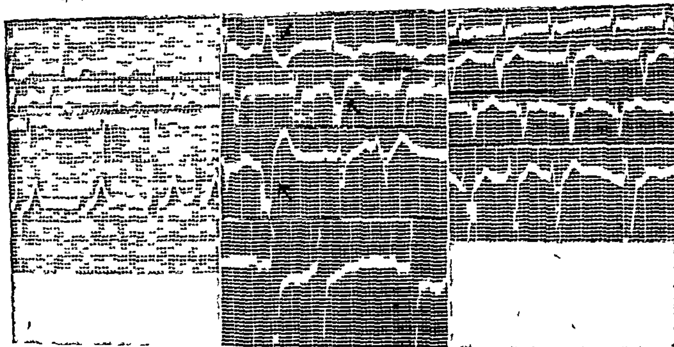
His condition was characterized by intermittent periods of pulmonary congestion mental confusion cyanosis and dyspnea

Autopsy—Extensive arteriosclerosis of the coronary arteries and aorta thrombosis of anterior descending branch of left coronary artery large recent organizing infarct apex of the left ventricle large mural thrombus of the left ventricle and left auricular appendage Hypertrophy of cardiac chambers especially the left ventricle the wall of the left ventricle in the region of the infarct is not over 8 to 10 millimeters in thickness in contrast to the basal portion which reaches a maximum of 20 millimeters

Thrombosis of periprostatic veins bilateral pulmonary embolism thrombosis with hemorrhagic infarcts of right lower lobe hyperemia and edema of lungs bilateral hydrothorax (right 500 cc and left 200 cc) Chronic passive hyperemia of the lungs liver spleen and kidneys

Summary—A case of recent anterior myocardial infarct is presented with hypertension and auricular fibrillation present prior to infarction Death due to advanced cardiac failure and pulmonary embolism thrombosis with pulmonary infarcts probably from thrombosis of periprostatic veins

PLATE 38 RECENT ANTERIOR MYOCARDIAL INFARCT WITH CARDIAC FAILURE



T diphasic

Ventricular premature systoles T_1 deeply inverted coved ST depressed T isoelectric

QRS_1 low QRS_4 inverted T_1 iso-electric T low

Auricular fibrillation and left axis deviation present in all three tracings Changes in Leads 1 and 4 (QRS ST T) on various dates indicate recent anterior myocardial infarct Auricular fibrillation was present before the infarction



Left ventricle opened from the front showing aorta and aortic valve Mitral valve to the right Large recent organizing infarct apex of the left ventricle



Left ventricle opened from the back showing left auricle mitral valve *Mural thrombus* of left ventricle and left auricular appendage

The wall of the left ventricle in infarcted area is only 8 mm thick at the base 20 mm left ventricular hypertrophy due to associated hypertension (left axis deviation in electrocardiogram)

PLATE 39 OLD CALCIFIED ANTERIOR INFARCT WITH VENTRICULAR ANEURYSM

A B, male, white, aged 66 *Admitted on March 17, 1942, died on March 21, 1942*

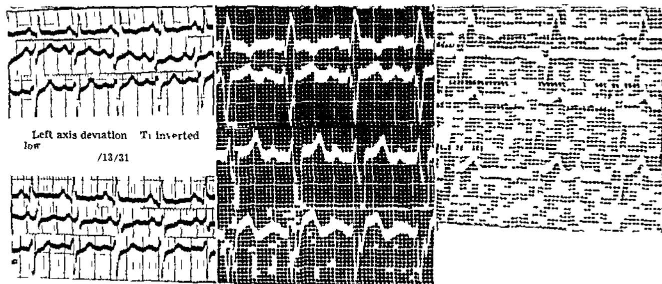
Patient had a coronary attack thirteen years prior to admission at 53 years of age, and remained in bed for a month He weighed 210 pounds at the time but gradually reduced to 165 pounds Ten years later he developed diabetes mellitus A year before admission he began to have attacks of nocturnal dyspnea as a sign of advanced left ventricular failure, but no precordial pain The examination made at that time revealed heart regular rhythm, no murmurs liver enlarged with fluid in abdomen, edema of lower extremities Blood pressure 140/70 Urine albumin 1 to 3 plus no sugar occasional red cells a few hyaline granular casts He felt well until his last admission with acute bronchopneumonia (March 17 1942) he died four days later

Autopsy—Old healed occlusion of the anterior descending branch of the *left coronary artery old, healed infarction with parietal aneurysm mural* thrombosis and calcification of the wall of the left ventricle Chronic dilatation and disseminated calcification of the mitral ring Chronic passive hyperemia *of the lungs liver (cyanotic induration) spleen and kidneys* Acute bronchopneumonia Fat infiltration of the pancreas (clinical diagnosis of diabetes mellitus) Slight benign nephrosclerosis

Summary—A case of an old anterior myocardial infarct with a large ventricular aneurysm is presented The large portion of the wall of the aneurysm consisted of bone and calcified scar tissue This change developed over a period of thirteen years Death was due to intercurrent broncho pneumonia

PLATE 39 OLD CALCIFIED ANTERIOR INFARCT WITH VENTRICULAR ANEURYSM

2/2/31 Aged 55 | 4/3/41 Aged 65 | 1/[illegible]/4[illegible] Aged 6[illegible]



The persistent changes in Lead 1 (Q_1 present T inverted) and Lead 4 (QRS_4 inverted ST_4 slightly elevated and deformed) suggest an old healed anterior infarct with ventricular aneurysm The *persistent left* bundle branch block suggests involvement of the interventricular septum



?/14 31 Wide aorta

Ventricular aneurysm of anterior wall and septum consisting of bone and calcified scar tissue developed over a period of thirteen years

PLATE 40 DISSECTING ANEURYSM OF THE LEFT CORONARY ARTERY OLD ANTERIOR INFARCT WITH PERFORATED SEPTUM

P B, male, white aged 71 Admitted on June 2, 1942, died on June 29, 1942

Patient had been in good health when, on the day of admission, he was awakened with severe precordial and epigastric pain, which radiated bilaterally to the back and scapular regions and down both arms to the elbows he broke out into a cold sweat The pain continued without abating for several hours and in the morning he vomited some food He had never had any pains previously He had had hypertension (over 200) for a few years

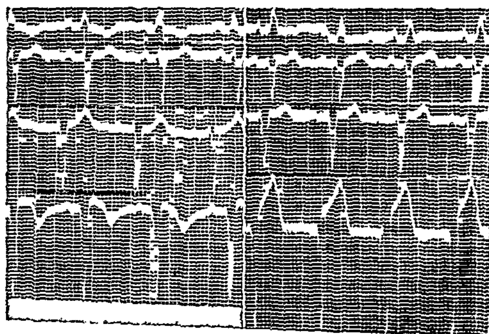
The heart was enlarged to the left and right the tones were distant no murmurs blood pressure, 138/94 Abdomen distended White count 19 000 On the eighth day the patient developed pulmonary infarction and ascites He died suddenly on the twenty eighth day

Autopsy—Generalized and marked coronary arteriosclerosis but no thrombi Dissecting aneurysm of the anterior descending branch of left coronary artery, causing its obstruction Old healed infarct of the anterior wall of the left ventricle and ventricular septum with parietal aneurysm The interventricular septum is exceptionally thin and there is a parietal aneurysm of the septum bulging from left to right side In the central portion there is an ovoid septal defect the edges are thickened smooth firm pearly white suggesting that they are made of thickened tissue and that defect has been present for a long time Apical mural thrombus of left ventricle Dilatation and hypertrophy (20 mm) of the left ventricle Acute hemorrhagic (aspiration) bronchopneumonia Pulmonary embolic thromboses with hemorrhagic infarct of right lower lobe Bilateral hydrothorax ascites and general anasarca due to cardiac failure

Summary—A case of dissecting aneurysm of descending branch of left coronary is presented superimposed upon an asymptomatic old healed anterior infarct with aneurysm and perforated interventricular *septum* *Death* due to cardiac failure bronchopneumonia and pulmonary embolism thromboses with infarct

PLATE 40 DISSECTING ANEURYSM OF THE LEFT CORONARY ARTERY OLD ANTERIOR INFARCT WITH PERFORATED SEPTUM

6/3/4? Aged 41 | 6/9/4?



Left axis deviation QRS low QRS₄ inverted T_1 4 inverted coved

T_1 low inverted ST_4 markedly elevated deformed fused with T_4

The changes noted in Leads 1 and 4 especially in ST_4 and T_4 indicate active recent anterior coronary disease



6 0 4 *Heart* enlarged to the left and right as in cardiac failure with dilatation of both ventricles Bronchopneumonia



Old healed anterior myocardial infarct Ventricular aneurysm Old *healed* perforated interventricular septum defect

PLATE 41 RECENT POSTERIOR INFARCT WITH ACUTE PERICARDITIS

P R, male, white aged 63 Admitted on Jan 2 1942, died on Jan 15 1942

Two and one half months previous to admission the patient had an attack of precordial pain, accompanied by dyspnea Five weeks later, the patient developed a substernal pain which was different than he had experienced previously, the pain did not radiate to the left arm or neck, and was not related to food taken or exercise

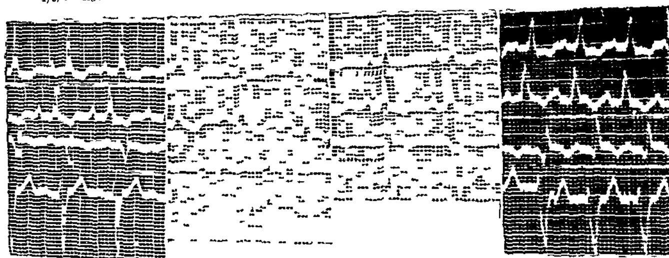
On admission pulse 86, temperature, 101 8° F, blood pressure, 128/78 The heart sounds were distant, regular, no murmurs The liver was two fingerbreadths below the costal margin, slight pitting ankle edema White count, 21,600 Four days later there was a definite to and fro friction rub over the lower precordium One day before death, patient complained of pain in precordium and left shoulder, he was apprehensive, his color was ashen there were rales in posterior base of the lungs, pulse 130 to 145 Blood pressure 130/105 Patient died next morning

Autopsy—Atherosclerosis of the aorta and coronary arteries with calcification and ulceration Recent occlusion of the circumflex branch of the left coronary artery organizing recent myocardial infarction, posterior wall of the left ventricle near base organizing acute sanguinoserofibrinous pericarditis hypertrophy and dilatation of the left and right ventricles and dilatation of right auricle Chronic passive hyperemia of the lungs liver, spleen and kidneys Moderate bilateral hydrothorax compression atelectasis of both lower pulmonary lobes Organizing infarct of the spleen arteriosclerotic atrophy of the kidneys

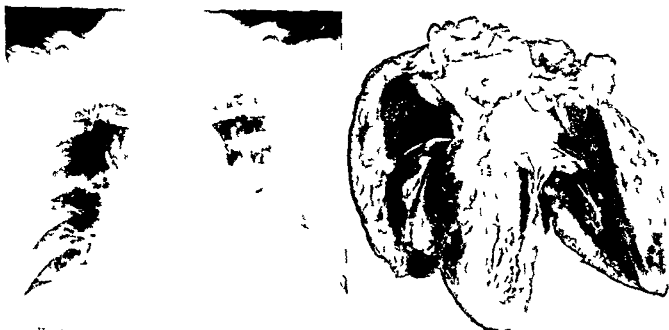
Summary—A case of a recent posterior myocardial infarct with acute pericarditis and cardiac failure is presented Although the infarct was posterior a pericardial rub was heard anteriorly due to generalized pericarditis

PLATE 41 RECENT POSTERIOR INFARCT WITH ACUTE PERICARDITIS

1/3/4 Aged 63 1/"/4° 1/8/4° 1/13/4°



The ele trocardiographic changes in Leads 2 and (Q3 present, high take-off of ST3 T 3 inverted) suggest a recent posterio *infarct in* addition to active anterior coronary disease (T1 low diphasic or inverted QRS4 inverted or low)



Hea t markedly enlarged in all directions due to peri carditis and cardiac failure

Recent posterior myocardial infarct Acute san guinoserofibrinous pericarditis

PLATE 42 OLD POSTERIOR INFARCT WITH VENTRICULAR ANEURYSM

I B male, white, aged 61 Admitted on March 14, 1935, died on May 5, 1935

The patient had been in ill health periodically for twelve years Nine years before admission he had been told that he had angina pectoris In the last six years, he had had dyspnea, which was his chief complaint on admission On physical examination the heart was enlarged to the left the rate regular, no murmurs there was cyanosis of the lips, pulsation of the veins of the neck, dyspnea Blood pressure 190/120 After bed rest, blood pressure fell to 130/70

On further questioning, patient stated that in the past four years he had begun to manifest some signs of cerebral vascular disturbance with mental instability, most marked in the past year and with periods of irrationality and excitement The patient died quite suddenly on May 5, while talking to the attending physician The death was ascribed to a cerebral accident

Autopsy—Marked generalized arteriosclerosis, arteriosclerotic occlusion of the right coronary artery Old myocardial infarct and parietal aneurysm of the posterior wall of the left ventricle and interventricular septum localized fibrous pericarditis mural thrombosis hypertrophy of both cardiac ventricles Old dissecting aneurysm of the right common iliac artery

Arteriosclerotic atrophy of the kidneys healing infarcts of the kidneys *Acute distention of the lungs*

No permit for autopsy of the brain could be obtained

Summary—A case of an old posterior myocardial infarct with a ventricular aneurysm is presented Death was probably due to a cerebral accident

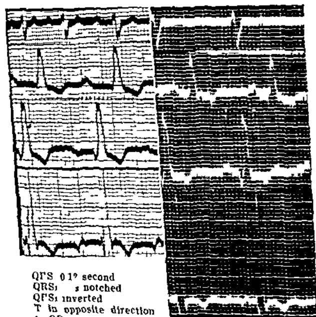
PLATE 42 OLD POSTERIOR INFARCT WITH VENTRICULAR ANEURYSM



9/15/3? Left ventricular hypertrophy due to hyper tension Left border is rounded and out

3/15/35 Marked left ventricular hypertrophy and dilatation.

3/30/35 Aged 61 4/10/35 B P 190/1?0



QRS 0 1? second
QRS₁ ₃ notched
QRS₁ inverted
T in opposite direction to QRS

Similar to 3/30/3

Right bundle branch block ST ₃ depressed deformed T ₃ inverted Posterior myocardial infarct of the base of left ventricle with septum involvement (right bundle branch block)



Old posterior infarct with ventricular aneurysm of posterior wall of the left ventricle base and septum

PLATE 43 RECENT ANTERIOR AND POSTERIOR INFARCTS

C W, white, female, aged 64 Admitted on April 20, 1939 died on April 28 1939

History of hypertension (200 to 220) in previous years with several attacks of epileptiform convulsions due to cerebral arteriosclerosis Nine months before admission there was a hacking cough which persisted for five months The roentgenogram of the chest revealed a markedly dilated heart Patient was relatively well until nine days before admission when she had a coughing spell with marked perspiration coolness and pallor with blood pressure below 100

On admission marked dyspnea cough cyanosis of lips and hands Pulse 70, temperature 99 4 F, respirations, 26 blood pressure 164/90 The apex is diffuse, in anterior axillary line premature systoles, apical systolic murmur Right pleural effusion Liver not enlarged no ascites no ankle edema

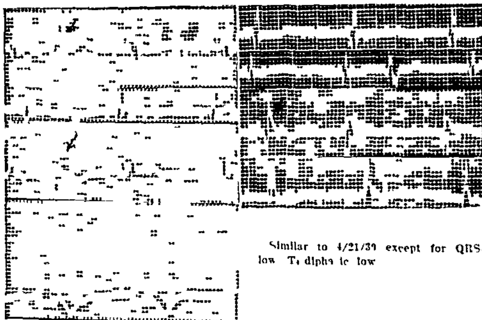
Her condition became serious five days after admission because of marked dyspnea, cyanosis, and stupor temperature rose to 102 4° F

Autopsy—Generalized arteriosclerosis marked sclerosis of coronary arteries, recent thrombosis of the descending branch of left coronary and recent dissecting aneurysm of right coronary artery recent infarction of the anterior and posterior walls of the left ventricle and ventricular septum organizing mural thrombi of the left ventricle and auricle dilatation of both ventricles and auricles hypertrophy of right ventricle Numerous organizing thrombi of the pulmonary artery hemorrhagic infarcts of the lungs right hydrothorax chronic compression atelectasis of right lung bronchiectasis sclerosis of the pulmonary arteries

Summary—A case of recent anterior and posterior infarcts discovered at autopsy is presented Patient was admitted with a chronic respiratory infection and died of pulmonary infarcts

PLATE 43 RECENT ANTERIOR AND POSTERIOR INFARCTS

4/21/39 Aged 64 4 2?/39 B P 164/90



Similar to 4/21/39 except for QRS₁ low T₁ diphasic low

Regular rhythm except for occasional ventricular premature systole and absent P suggest nodal rhythm or auricular fibrillation with complete auriculoventricular heart block Low voltage of all limb leads (QRS T) suggests myocardial (coronary) insufficiency



Heart shadow increased in all directions as in cardiac failure
Right hydrothorax

Recent anterior and posterior infarcts
Large thrombus in the left auricle

PLATE 44 RECENT ANTERIOR AND OLD POSTERIOR INFARCTS WITH CARDIAC FAILURE

H S male, white aged 51 Admitted on Nov 23 1941 died on Dec 4 1941

Patient was well until eighteen months before admission at which time he began having pains in the right upper quadrant with tiredness and weakness He grew progressively worse and developed dyspnea on exertion and edema of the ankles Eight months before admission he was put on digitalis he felt better and was able to carry on light work for two months then he suffered a relapse with dyspnea on slightest activity with fluid in abdomen and ankle edema He gradually improved and felt well until six weeks before admission when the edema dyspnea and weakness returned

On admission pulse 90 temperature 98 4° F respirations 24 blood pressure 120/80 Heart regular rhythm no murmurs the liver was palpated two fingerbreadths below the right costal margin with some tenderness fluid in abdomen edema of the lower extremities no dyspnea while in bed no cyanosis He grew gradually worse with a steady increase in temperature pulse respiration and cyanosis He became irrational and delirious

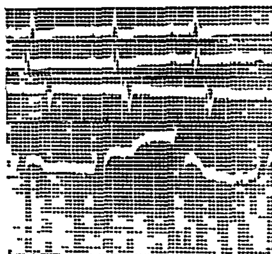
Autopsy—Recent occlusion of the anterior descending branch of the left coronary artery with an organizing infarct of the anterior portion of the ventricular septum and left ventricle with a mural thrombus Old healed occlusion of the circumflex branch of the left coronary with a healed infarct of the posterior portion of the left ventricle and ventricular septum at the base, with a parietal aneurysm Dilatation of the left ventricle hypertrophy of the right ventricle chronic dilatation of the right heart chambers Marked bilateral hydrothorax ascites chronic passive hyperemia of abdominal viscera *Acute bronchopneumonia Emboli thromboses of branches of the pulmonary artery* to the right upper lobe with hemorrhagic infarct

Summary—A case of recent anterior and old posterior infarcts with advanced heart failure is presented Death due to pulmonary infarct

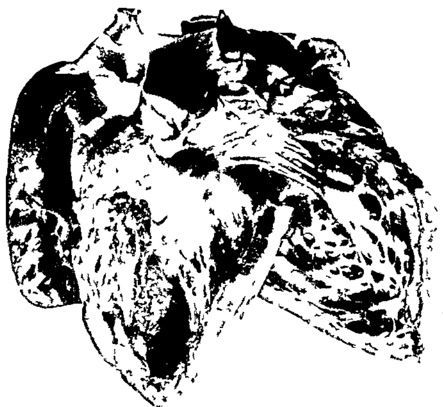
PLATE 44 RECENT ANTERIOR AND OLD POSTERIOR INFARCTS WITH CARDIAC FAILURE

11/28/41 Heart markedly enlarged as in advanced cardiac failure Bilateral bronchopneumonia

11/25/41 Aged 51 Rate [illegible]4



Left axis deviation Leads 1 2 3 (QRS T) of low voltage myocardial insufficiency QRS₄ inverted T₄ low diphasic coronary disease Prolonged PR—0 24 second Incomplete auriculoventricular block suggests ventricular septum involvement



Recent anterior infarct with mural thrombus Old healed posterior infarct with ventricular aneurysm Dilated left ventricle (cardiac failure)

PLATE 45 ANTERIOR MYOCARDIAL INFARCT

The infarct is in the anterior wall, apex of the left ventricle, and anterior portion of the interventricular septum It results usually from occlusion of the descending branch of the left coronary artery, supplying the above region

In acute anterior infarction, the electrocardiographic changes occur in Lead 1, Q_1T_1 pattern QRS_1 low voltage, Q_1 prominent (while S_3 reciprocally deep) ST_1 elevated high take off, and fused with T_1 (while ST_3 reciprocally depressed and fused with T_3) Leads 2 and 4 usually show changes similar to Lead 1, as Lead 4 is the apical precordial lead and in close proximity to the infarcted area

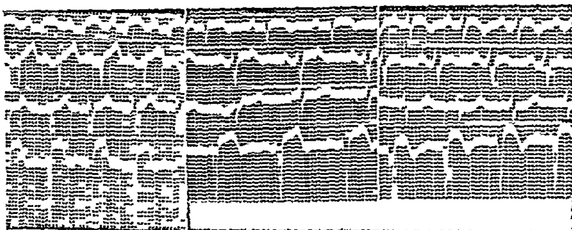
It may take a few hours to a day for the above changes to appear in an acute coronary attack, and it is not unusual to find a normal electrocardiogram (normal conduction) in the first few hours of an acute attack

In a few days while myocardial infarct shows signs of healing myocardial conduction improves and some of the electrocardiographic changes gradually return to normal ST returns to base line but $T_{1\ 4}$ become deeply inverted coved and QRS_1 inverted (normally diphasic)

The electrocardiographic changes of healing myocardial infarct (inverted T) may persist for a few weeks and gradually return to normal in which case a small myocardial scar (fibrosis) remains as the only result of a healed myocardial infarct If the scar (fibrosis) is extensive or as frequently happens a ventricular aneurysm develops then the ventricular conduction is permanently impaired and the electrocardiographic changes of a healing myocardial infarct with inverted T remain for years If the electrocardiograms taken at various dates over a long period of time show persistent changes a ventricular aneurysm should be strongly suspected This condition often causes a myocardial (coronary) insufficiency with left ventricular failure and dyspnea on exertion

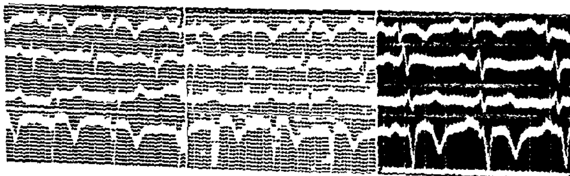
PLATE 45 ANTERIOR MYOCARDIAL INFARCT WITH CHANGES IN LEADS 1 AND 4

3 14/45 Aged 48 3/21/45 3/ ? 45



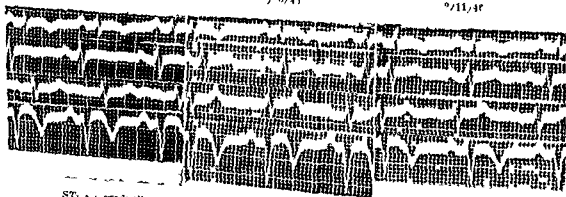
Left axis deviation suggests left ventricular hypertrophy. QRS_1 low inverted. QRS inverted. ST_1, 4 elevated fused with T_1, 4. *Above ECG* changes in Leads 1, 4 suggest *active* impairment in myocardial conduction due to acute infarction in anterior wall apex of the left ventricle

4/9/4 4 16/4 5/ 1/45



ST back to base line and convex now. T_1, 4 inverted coved. Above ECG changes suggest improvement in *myocardial* conduction and healing of the infarction

? 7/45 / 0/45 ?/11/46



ST_1, 2, 4 gradually became a normal base line. T_1 low inverted not coved. T isoelectric first then up. T_4 deeply inverted coved.

Above changes over period of eleven months suggest a gradual improvement in myocardial conduction. If inversion of QRS and T remain permanently unchanged a development of ventricular aneurysm in an old healed infarcted area is to be considered

Electrocardiogram taken on 10/1 /46 is similar to /11/46. No changes

PLATE 46 POSTERIOR MYOCARDIAL INFARCT

The infarct is in the posterior wall, base of the left ventricle and posterior portion of the interventricular septum It usually results from occlusion of the circumflex branch of the left coronary or of the right coronary artery, supplying the above named region

In acute posterior infarction the electrocardiographic changes occur in Leads 2 and 3, Q_3T_3 pattern QRS low Q 3 prominent (while Q_1 absent) ST_3 elevated and fused with T_3 (while ST_1 reciprocally depressed and fused with T_1)

In a few days while myocardial infarct shows signs of healing, the myocardial *conduction improves and some of the electrocardiographic changes* gradually return to normal ST returns to base line but T 3 become deeply inverted

The electrocardiographic changes of healing infarct (inverted T 3) may persist for a few weeks and gradually return to normal, or remain as such unchanged for months or years suggesting a ventricular aneurysm

Case 1 M C female white aged 71

Acute posterior infarct with rupture of the heart

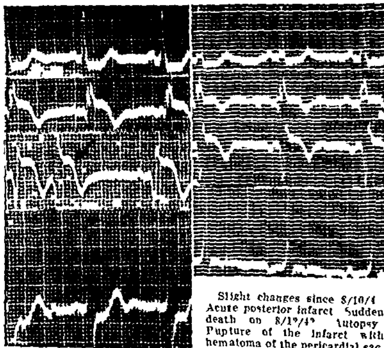
Hypertension for fifteen years In the last year intermittent attacks of vicelike substernal pain on exertion or occasionally after eating radiating down both arms relieved by rest—coronary disease The evening before admission acute attack of sharp precordial pain nausea and vomiting no dyspnea Blood pressure 206/100 Heart regular no murmurs Fever 99 to 102 4° F Leucocyte count 13 600 On the fourth day the blood pressure was 168/88 and patient died rather suddenly

Autopsy—Marked aortic and coronary arteriosclerosis Acute occlusion of the posterior descending branch of the right coronary artery due to hemorrhage into an arteriosclerotic plaque Acute infarction of the posterior wall of the left ventricle Spontaneous rupture of the infarct with hematoma of the pericardial sac

PLATE 46 POSTEPIOR MYOCARDIAL INFARCT WITH CHANGES IN LEADS 2 AND 3

CASF 1

M C aged 1 8/10/4 8/1°/4

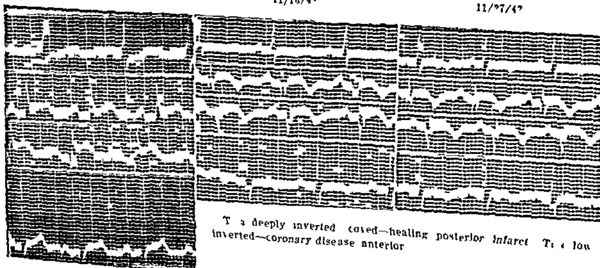


Q prominen ST_2 s high take-off T s inverted Premature systole (Lead 3)

Slight changes since 8/10/4 Acute posterior infarct Sudden death on 8/1°/4° Autopsy Pupture of the infarct with hematoma of the pericardial sac

CASE °

A P aged 80 10/30/4° 11/16/4° 11/°7/4°



T_3^{ST} high take-off fused with

T s deeply inverted coved—healing posterior infarct T, s low inverted—coronary disease anterior

Two weeks after admission patient develop d acute thrombophlebitis of left femoral vein followed later by pulmonary embolism and death on 1 /3/4°

Autopsy Death due not to the posterior myocardial infarct but to pulmonary embolism from thrombosis of the l ft femoral vein

PLATE 47 MYOCARDIAL INFARCTION WITH PAROXYSMAL AURICULAR FLUTTER

In myocardial infarct, especially when extensive, the unaffected portion of the left ventricular myocardium may fail, the myocardial insufficiency in this case being the result of a marked coronary insufficiency. This is evidenced in the electrocardiogram by marked left axis deviation, low voltage of all the leads (QRS, T) or the ST's in Leads 1 and 4 or 2 and 3, instead of high take off or elevation may be markedly depressed, concave, and fused with T's. Clinically there will be dyspnea as the result of the left ventricular failure.

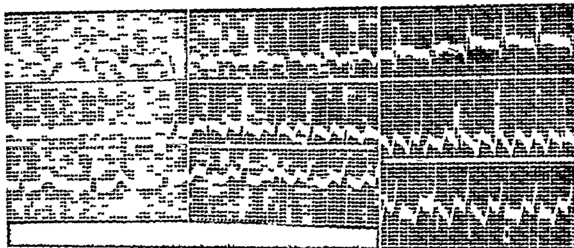
In other cases the myocardial infarct may be the focus for cardiac irritability and cause appearance of premature systoles or paroxysmal auricular flutter as described in the following case.

D. I., male, white, aged 58, hypertension for the last few years. Three days before the first electrocardiogram was taken patient had his first coronary attack, with sharp precordial pain, fever, leucocytosis and blood pressure drop from 240/120 to 120/80. Two days later patient had a sudden onset of paroxysmal auricular flutter 2:1 with auricular rate of 352 and ventricular 176. Digitalis one cat unit (0.1 Gm.) four times daily, changed the regular ratio (2:1) into irregular (4:3, 2:1) while quinidine sulfate 0.2 Gm. every six hours re-established the normal sinus rhythm. Exactly a year later a second coronary attack with sharp precordial pain, fever, leucocytosis and marked drop in blood pressure recurred with reappearance of *paroxysmal 2:1 auricular flutter. Quinidine re-established a normal sinus* rhythm next day. Patient was well for two years when the third coronary attack occurred with sudden death.

Summary.—A case of myocardial infarction with paroxysmal auricular flutter *is presented*.

PLATE 47 MYOCARDIAL INFARCTION WITH PAROXYSMAL AURICULAR FLUTTER

6/8/35 Aged 58 B P 240/1 0 — 6/10/35 B P 120/80 — 6/13 35 Rate 146 to 176

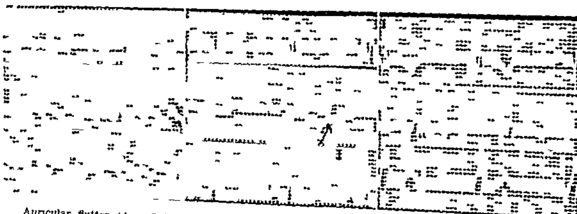


Q₁ pre ent T inverted coved T inverted low

Auricular flutter (2 1) Rate 1 6 ST₁ elevated Acute anterior infarct

Auricular flutter (3 or 2 1) Digitalis given

6/1 35 — 6/20/ 5 — 6 /3

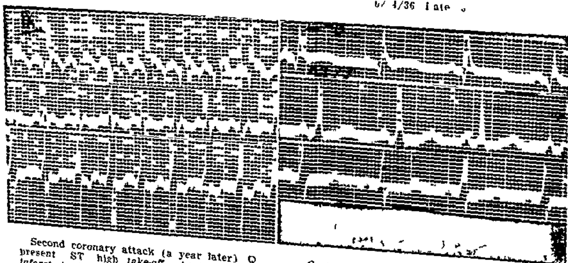


Auricular flutter (4 or 2 1) Quinidine given

Regular sinus rhythm Occasional auricular premature systole

Simi ar to 6/8/3

6/23 36 Rate 150 — 6/ 4/36 Rate



Second coronary attack (a year later) Q present ST high take-off Acute anterior infarct Auricular flutter (2 1) Quinidine given

Q₁ present ST₁ near base line suggesting improved conduction Regular sinus rhythm

PLATE 48 CORONARY DISEASE WITH A COMPLETE AURICULOVENTRICULAR HEART BLOCK

A. W., male white aged 56. Died on Nov. 9, 1945.

While in good health until July, 1939, patient developed a sudden attack of dyspnea without precordial pain, diagnosed as a coronary attack, and he was hospitalized for ten weeks. He felt well until Nov. 9 1945, when while punching the time clock at work *he dropped dead. No autopsy* was performed.

Electrocardiograms taken at various times between July 1939 and August, 1945 showed a gradual development of auriculoventricular block from a prolonged PR and occasional dropped beat in July 1939 to a com plete block *in May 1941 with a persistence of a complete block until death in* November 1945.

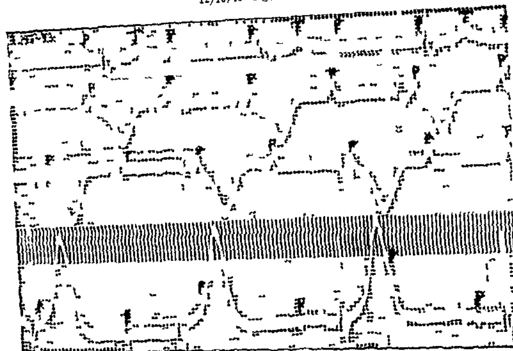
The presence of a complete auriculoventricular block indicated dis turbance in conduction in the His bundle and septum involvement in addi tion to that of ventricular myocardium proper (inverted T_3). The striking *feature in this case is the unusual high voltage of $T_{3,4}$ found accidentally* once on Dec. 13 1943 while patient was feeling fine and the electrocardio grams taken prior to and after this did not show this abnormality.

Summary.—A case of a coronary disease with a complete heart block is presented. Unusual high voltage of $T_{3,4}$ was once noted.

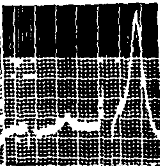
In complete auriculoventricular heart block no impulses reach the ven tricles from the auricles. The ventricles respond to a center situated below the block either in the His bundle with QRS and T normal in form and shape or in the ventricles proper with markedly aberrant QRS and T. The aberrant ventricular complexes are similar to those in bundle branch block or ventricular premature systoles. For comparison a single premature systole with high and aberrant T in Lead 4 of another case is shown and similarity with T_4 of above cited case is noted. Therefore unusually high $T_{3,4}$ *is not an indication of myocardial impairment* but due to abnormal initiation and conduction of ventricular impulses in complete auriculoven tricular heart block.

PLATE 48 CORONARY DISEASE WITH A COMPLETE AURICULOVENTRICULAR HEART BLOCK

12/15/43 Aged 54



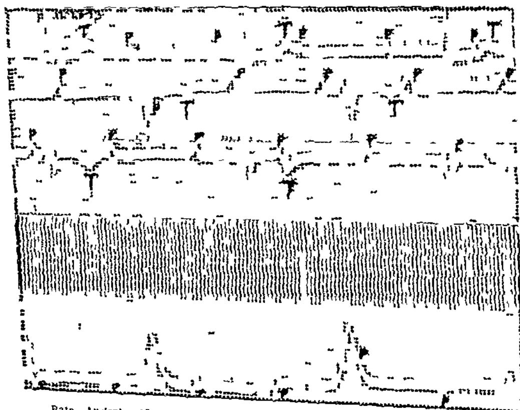
S S female white aged 53



Rate Auricular 68 ventricular 36 Complete heart block T_1 up (mm) T_2 deeply inverted (12 mm) T_4 up (25 mm) Posterior coronary disease No complaints Patient ambulant

Lead 4 Single ventricular premature systole with high T similar to T_4 in this ECG

12/28/43 The same patient



Rate Auricular 68 ventricular 0 Complete heart block Note the striking changes which occurred within two weeks No complaints Patient ambulant

IV

HYPERTENSIVE HEART DISEASE

Hypertension is only a symptom and not a disease entity There are many causes for hypertension, some of which are still unknown A thorough examination of the patient with complete correlation of history, physical findings, including eye ground examination, and various laboratory procedures, as roentgenograms, electrocardiograms, blood chemistry, urinalyses, and kidney function tests, may help the clinician to arrive at certain diagnostic conclusions, which would be of great importance for the treatment of the patient's *hypertension*

In hypertension, i e, increased tension in peripheral arteries, the *peripheral resistance, i e, diastolic pressure is high To overcome* the high peripheral resistance, the power of the heart, i e systolic pressure, must be increased This will manifest itself also by hypertrophy of the left ventricle, as shown on the roentgenogram and the electrocardiogram with left axis deviation or left ventricular strain The *high systolic pressure and the left ventricular hypertrophy are compensatory* mechanisms for high peripheral resistance The hypertrophy reaches a certain level, beyond which it cannot go and dilatation of the left ventricle sets in, with myocardial insufficiency, dyspnea, and left cardiac failure as physiological and clinical evi*dence of it* At this stage of cardiac failure and myocardial insufficiency in hypertension we are dealing with hypertensive cardiovascular disease If unrelieved, the left ventricular failure with dyspnea will be followed by that of the right, with cyanosis peripheral edema ascites, enlarged liver, hydrothorax, and, finally death

The kidney plays an important role, and glomerulonephritis is one of the causes of hypertension In estimating the anatomical and physiological impairment of the kidneys, the physician should not be satisfied with urinalysis and findings of albumin and casts or estimating the albumin globulin ratio and retention of nitrogenous substances in the blood but should perform kidney function tests

such as Mosenthal's concentration urea clearance, dye excretion phenolsulfonphthalein (PSP) to find out if there is any kidney insufficiency. If there is, and associated with myocardial insufficiency as above, we are dealing with cardiovascular renal disease.

Hypertension is frequently associated with arteriosclerosis of peripheral blood vessels including renal. This nephrosclerosis may cause traces of albumin and occasional hyaline casts in the urine but otherwise it is asymptomatic and should not be confused with glomerulonephritis. In renal arteriosclerosis the intima of the blood vessels is affected showing atheromatous plaques and ulcerations but the arterial lumen is patent.

The arterioles of the kidneys, as in any other organ of the body, may show a marked concentric thickening of the media, the increase in thickness due chiefly to hyalinized fibrous tissue, so that in places the lumen is completely obliterated and cannot be identified. This **renal obliterative endarteritis** is associated with hypertension and may cause renal insufficiency, similar to myocardial insufficiency by coronary occlusion.

If endarteritis affects the pulmonary arteries, or if any pulmonary disease, as emphysema, asthma, fibroid tuberculosis, causes *obstruction in pulmonary circulation*, we are dealing with **pulmonary hypertension** in contradistinction to systemic hypertension in systemic blood vessels. To overcome the pulmonary hypertension the right ventricle hypertrophies and establishes *a condition known as* **cor pulmonale**. The right ventricle may fail *and* cause cardiac failure and death.

Dilatation of the pulmonary artery and its branches due to increased pressure can be shown on the roentgenogram (see Plate 56): bulging of the left cardiac border in the *region* of the pulmonary artery and increased hilar shadows due to dilated secondary branches of the pulmonary artery. *The right ventricular* hypertrophy will cause right axis deviation or if marked right ventricular strain in the electrocardiogram.

The systemic hypertension causes hypertrophy of the left ventricle while pulmonary hypertension that of the right. The condition can be recognized by changes on the roentgenogram while the electrocardiogram shows left or right axis deviation or ventricular strain.

PLATE 49 HYPERTENSIVE HEART DISEASE WITH LEFT AND RIGHT CARDIAC FAILURE

F. S., male, white, aged 56. Admitted on Nov. 14, 1938, died on Jan. 9, 1939.

Hypertension and severe attacks of dyspnea on exertion of four years' duration. For the last two years, he had increased dyspnea with more orthopnea and he had also noticed swelling of the ankles; he had never had any precordial pain. In the last year he was given mercurial diuretics *intravenously as needed, with some improvement of dyspnea and edema.*

Examination on admission revealed an obese, edematous, dyspneic, orthopneic man who was propped up in bed and sweating profusely; the skin *of the face was flushing. There was edema over the sacral and lumbar spines,* and slight edema of the scrotum. Few crackling rales over both bases of the lungs. Blood pressure 180/110, pulse 84, regular. The left heart border in the anterior axillary line. A₂ accentuated; no murmurs. The abdomen was obese and flaccid. The liver was four fingerbreadths below the costal arch, not tender. The diagnosis was hypertension with left cardiac hypertrophy and left and right cardiac failure. On January 9 at 9:30 P.M. he was sitting on the edge of his bed without respiratory *difficulty but confused mentally and* with slight hallucinations. At midnight the patient was found dead.

Autopsy.—Marked hypertrophy of the right and left ventricles. Slight acute and chronic passive hyperemia of the lungs; *marked passive hyperemia* of the liver, kidneys, spleen and gastrointestinal tract. Right hydrothorax; slight ascites; marked anasarca; edema of the brain. Moderate generalized *arteriosclerosis.*

Summary.—A case of hypertensive heart disease with left and right cardiac failure is presented. Death due to advanced heart failure.

PLATE 49 HYPERTENSIVE HEART DISEASE WITH LEFT AND RIGHT CARDIAC FAILURE



1/ 6 38 Markedly increa ed cardiac shadow as in cardiac failure Increased lung markings (cardiac)



Mark d hypertrophy of the left ventricle The heart with the aorta attached weighed 800 grams

PLATE 50 HYPERTENSION

Illustrating roentgenographic and electrocardiographic changes in hypertension with left ventricular hypertrophy.

Roentgenogram.—Compare with Plate 1 and note that a normal left ventricular border is straight while in left ventricular hypertrophy the border is rounded. The rounding of the border is due to elongation and hypertrophy of the muscle fibers and is the same in any left ventricular hypertrophy of whatever cause it may be: hypertension, aortic insufficiency or stenosis of rheumatic, arteriosclerotic or syphilitic etiology.

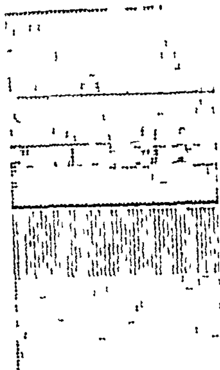
Compare also with Plate 12, anterior view.

Electrocardiogram.—The left ventricular hypertrophy manifests itself in the electrocardiogram in mild cases by left axis deviation and in marked cases by left ventricular strain: left axis deviation with high, wide, slurred QRS; ST_1 depressed and fused with inverted T_1 and often with similar changes in Lead 4. The left ventricular strain is an indication of a marked hypertrophy and not of a damaged myocardium. Dilatation and failure of the left ventricle with dyspnea may follow this stage of ventricular hypertrophy (see Plate 51).

J W male white aged 63

Left ventricular border is slightly rounded Some calcification of the aortic arch

Aged 63 BP – 0/110

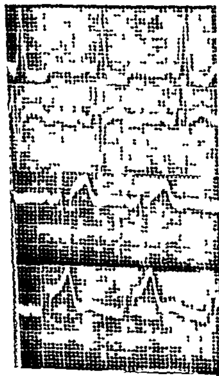


QRS inverted left axis deviation

J K male white aged 58

Left ventricular border is markedly *rounded* Marked hypertrophy of outflow tract left ventricle

Aged 58 00/110



Left ventricular strain
Left axis deviation QRS wide high slurred ST_1 depressed and fused with inverted T_1

PLATE 50. HYPERTENSION

Illustrating roentgenographic and electrocardiographic changes in hypertension with left ventricular hypertrophy.

Roentgenogram.—Compare with Plate 1 and note that a normal left ventricular border is straight while in left ventricular hypertrophy the border is rounded. The rounding of the border is due to elongation and hypertrophy of the muscle fibers and is the same in any left ventricular hypertrophy of whatever cause it may be: hypertension, aortic insufficiency or stenosis of rheumatic, arteriosclerotic or syphilitic etiology.

Compare also with Plate 12, anterior view.

Electrocardiogram.—The left ventricular hypertrophy manifests itself in the electrocardiogram in mild cases by left axis deviation and in marked cases by left ventricular strain: left axis deviation with high, wide, slurred QRS, ST_1 depressed and fused with inverted T_1 and often with similar changes in Lead 4. The left ventricular strain is an indication of a marked hypertrophy and not of a damaged myocardium. Dilatation and failure of the left ventricle with dyspnea may follow this stage of ventricular hypertrophy (see Plate 51).

PLATE 51 HYPERTENSION WITH LEFT VENTRICULAR FAILURE AND ARTERIOSCLEROSIS



Left ventricular hypertrophy and dilatation Wide aorta Left cardiac border is rounded and out

Left oblique view Left ventricular border over lapping the spine Diffusely dilated aorta

4/7/43 Aged 60 B P 180/110 3/3/44 1/ 0/4[illegible]

Left ventricular strain Coronary dis[illegible]

PLATE 51 HYPERTENSION WITH LEFT VENTRICULAR FAILURE AND ARTERIOSCLEROSIS

S F, male, white, aged 62

Attacks of nocturnal dyspnea due to left ventricular failure, in the last two and one half years occurring about every three to four months Blood pressure, 180/110 Pulse, 80 regular except for ventricular premature systoles Heart markedly enlarged to left with wide aorta A₂ accentuated no murmurs heard Urine albumin 2 plus occasional casts Blood chemistry, normal

Roentgenogram, Anterior View—The left cardiac border is rounded and way out to the left due to hypertrophy and dilatation of the outflow tract of the left ventricle (anterior portion from apex to aortic valve) The aorta is diffusely enlarged the upper right cardiac border is bulging convex due to dilated ascending aorta, aortic knob is prominent and there is a lineal shadow descending from the aortic knob to the left of the pulmonary artery due to dilated descending aorta

Roentgenogram, Left Oblique View—*As the greater part of the left ventricle* is situated posteriorly and cannot be seen on the anterior view to visualize it the heart is rotated clockwise around the longitudinal axis placing the patient in left oblique position The spine is on the left side and normally the left ventricle inflow tract (posterior portion from mitral valve to apex) just touches the spine In this roentgenogram the left ventricle is markedly overlapping the spine thus visualizing the marked hypertrophy and dilatation of the left ventricle inflow tract in addition to that of outflow tract as seen in anterior view Note the diffusely and markedly dilated aorta due to arteriosclerosis

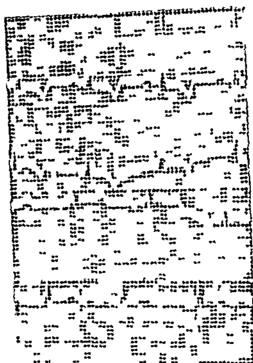
Summary—A case of hypertension with left ventricular failure attacks of nocturnal dyspnea and arteriosclerosis is presented to demonstrate characteristic roentgenographic changes in the anterior and left oblique views

PLATE 52 GLOMERULONEPHRITIS WITH HYPERTENSION AND UREMIC PERICARDITIS

3/9/38 B1 240/1 0



Left ventricular hypertrophy (mm thick) Pericarditi Slight athero clero is of the aortic arch



Left ventricular strain Left axis devi ation with high slurred QRS and inverted T_1 No hi tory of myocardial infarction



The surface of both kidneys is granular Section The cortex has undergone marked distortion and atrophy varying 3 to 5 mm in thickness Some distortion of pelvis Histology Chronic glomerulonephritis with a marked decrea e in number of glomeruli and a marked d gree of sclero is of the intralobular arteries

PLATE 52 GLOMERULONEPHRITIS WITH HYPERTENSION AND UREMIC PERICARDITIS

D. D., female, white, aged 43. Admitted on Feb. 20, 1938, died on March *31, 1938.*

Headaches and *dizziness of two years' duration.* Examination made three months before admission showed urine, 1 plus albumin, and occasional red cells; blood urea nitrogen, 57.8 mg. per cent; normal blood count; blood pressure 240/126. Blood pressure normal five years before admission.

On admission, the eye grounds were normal except for vascular tortuosity of the retinal vessels. Heart enlarged to the left, apex beat forceful, rate, 90, regular; blowing systolic murmur heard over the entire precordium but maximal in mitral area. A2 loud and snappy. Blood pressure 240/120. Electrocardiogram: left ventricular strain. Urine: specific gravity 1.014, albumin 3 plus, few erythrocytes. *Blood: hemoglobin 43* per cent, *red cells 2,300,000* (anemia); white cells 8,000. Urea nitrogen 87.4, uric acid 7.9, creatinine 6.2, nonprotein nitrogen 168.5, thus indicating a retention in the blood of *renal excretory substances. Carbon dioxide combining power decreased to* 40.7 (altered acid-base balance). Chlorides 544. Phenolsulfonphthalein (P.S.P.) *test returned only 10 per cent in one and one half hours (decreased excretion of dye).*

Patient's course in the hospital was characterized by nausea and vomiting, scanty urinary output, high nitrogen in the blood. Three weeks after admission a precordial friction rub was heard for several days: uremic pericarditis. Her condition continued slowly downhill.

Autopsy.—Chronic glomerulonephritis. Left ventricular hypertrophy (22 mm. in thickness). Anasarca; bilateral hydrothorax; ascites. Chronic passive hyperemia of the liver and spleen. Moderate arteriosclerosis of abdominal aorta, considerably less in the root and arch. Slight coronary sclerosis. Small organizing myocardial infarcts. Slight acute myocarditis; acute serofibrinohemorrhagic pericarditis. Bronchopneumonia.

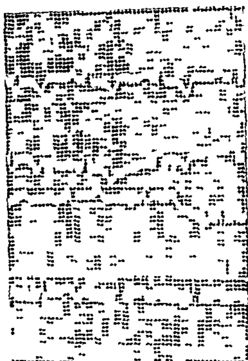
Summary.—A case of glomerulonephritis with hypertension and uremic pericarditis is presented. Death due to renal insufficiency (uremia).

PLATE 52 GLOMERULONEPHRITIS WITH HYPERTENSION AND UREMIC PERICARDITIS

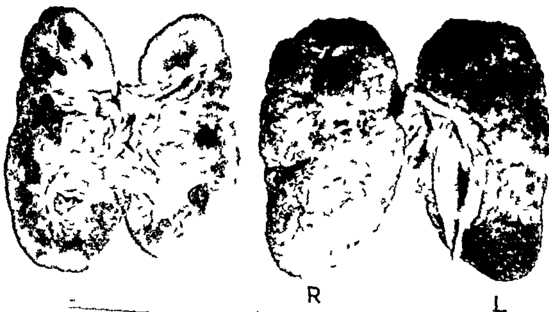


Left ventricular hypertrophy (mm thick) Pericarditi Slight atherosclerosis of the aortic arch

3/9/38 BH 40/1 0



Left ventricular strain Left axis deviation with high slurred QRS and inverted T₁ No history of myocardial infarction



The surface of both kidneys is granular Section The cortex has undergone marked distortion and atrophy varying 3 to 5 mm in thickness Some distortion of pelvis Histology Chronic glomerulonephritis with a marked decrease in number of glomeruli and a marked degree of sclerosis of the intralobular *arteries*

PLATE 53 REACTIVATION OF GLOMERULONEPHRITIS WITH HYPERTENSION

D. D., male, white, aged 36. Admitted on April 3, 1940; died on April 16, 1940.

Pneumonia in November, 1925, followed by nephritis. He felt well until a few months before admission when he began having severe headaches and nose bleed. Two weeks before admission he noticed impaired vision in his left eye. On admission blood pressure 228/128, heart enlarged to left; soft blowing apical murmur due to anemia, A_2 equal to P_2. Red cell count, 2 500 000; hemoglobin, 51 per cent; urine specific gravity 1 008, alkaline, albumin 4 plus, 120 red cells, 2 granular casts per cubic millimeter. Urea nitrogen, 53; total nonprotein nitrogen, 70.

Fundi.—Bilateral hypertensive neuroretinopathy with narrowing of retinal arterioles and arteries. Numerous small hemorrhages. The anemic pallor of the discs, opaqueness of retinae, fluffy white exudates would suggest a nephritic origin of the eye ground lesions rather than a malignant hypertension.

A week after admission, patient complained of substernal discomfort and dyspnea while in bed; red cell count dropped to 2 000,000. Hemoglobin 25 per cent. He died rather suddenly of acute pulmonary edema.

Autopsy.—Subacute and chronic glomerulonephritis. Hypertrophy of the left ventricle. Moderate atherosclerosis of the aorta. Numerous spontaneous hemorrhages in various parts of the body. Acute hemorrhagic bronchopneumonia.

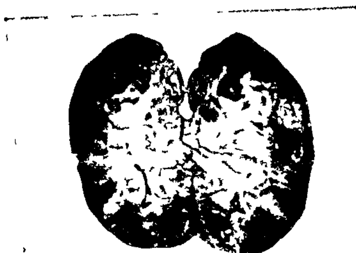
Summary.—A case of reactivation of glomerulonephritis is presented accompanied by hypertension and eye ground changes. Numerous spontaneous hemorrhages in various parts of the body with severe anemia. Death due to hemorrhagic bronchopneumonia.

PLATE 53 REACTIVATION OF GLOMERULONEPHRITIS WITH HYPERTENSION

D D male white aged 36 Blood pressure 228/128

The heart with the aorta weighs 609 grams The wall of the left ventricle is hyper trophied measuring 18 mm in thickness at the level of the mitral ring No changes in the valves endocardium myocardium or pericardium Lining of the auricles is smooth and gray Sections of myocardium are tan Moderate atherosclerosis of the aorta and coronary arteries





The initial kidney damage fifteen years ago was not very great since a relatively small number of glomeruli was the seat of a chronic stage

Exacerbation of a chronic glomerulonephritis in the last few months of life caused a subacute involvement of a great number of glomeruli

The degree of renal insufficiency was not very marked

PLATE 54 HYPERTENSION WITH BENIGN NEPHROSCLEROSIS

W B, male, white, aged 60 Admitted on Dec 20 1940, *died on Jan 21,* 1941

Dull headaches and tinnitus of two years duration Dyspnea daily for the past two months, with first attack of nocturnal dyspnea a few days before admission Blood pressure, 230/142 The heart enlarged to left, regular rhythm except for occasional premature systole, no murmurs No ankle edema *Red blood count, 3,500,000, hemoglobin 70 per cent* Urine slightly turbid, specific gravity, 1 012, albumin 4 plus, 30 white and 60 red blood cells per cubic millimeter 2 large granular casts nonprotein nitrogen 56 *chlorides, 425 The phenolsulfonphthalein test (P S P) revealed 25 per cent* excretion in one and one half hours

Three weeks after admission, patient developed bronchopneumonia Three days before death, nonprotein nitrogen, 83, creatinine, 2 9, carbon dioxide, 77 2, urea nitrogen, 68, chlorides, 440 On the last day, patient became comatose with pulse 132, temperature, 101 8° F abdomen markedly distended by ileus, marked pitting edema of both thighs

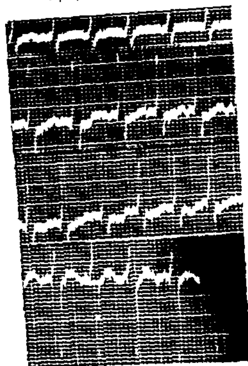
Autopsy—Generalized arteriosclerosis Marked benign nephrosclerosis Hypertrophy of the wall of the left ventricle Anasarca of the lower extremities Acute passive hyperemia of liver, stomach spleen and kidneys Acute hemorrhagic ulcerative colitis ileus Bronchopneumonia

Microscopic examination of the kidneys reveals advanced sclerosis of the intralobular and arcuate arteries rather than of the arterioles There are disseminated regions where atrophy of convoluted tubules and groups of glo*meruli with acute partial infarction and thrombosis are* separated by fairly well intact convoluted tubules and glomeruli

Summary—A case of hypertension arteriosclerosis with benign nephrosclerosis is presented Death due to bronchopneumonia

PLATE 54 HYPERTENSION WITH BENIGN NEPHROSCLEROSIS

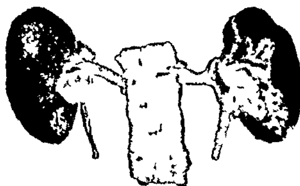
1 /23/40 B I 30/130



Lead I Paroxysmal auricular fibrillation



12/23/40 Wide descending aorta Left ventricular border is rounded Increased lung markings from hila down



Moderate atherosclerosis of the abdominal aorta Marked benign arteriosclerotic atrophy of the kidneys Renal arteries although altered by sclerosis have broadly patent lumina The surface of right kidney is granular The cut surface of left shows some alteration of the architecture of the cortex

PLATE 54. HYPERTENSION WITH BENIGN NEPHROSCLEROSIS

W. B., male, white, aged 60. Admitted on Dec. 20, 1940; died on Jan. 21, 1941.

Dull headaches and tinnitus of two years' duration. Dyspnea daily for the past two months, with first attack of nocturnal dyspnea a few days before admission. Blood pressure, 230/142. The heart enlarged to left, regular rhythm except for occasional premature systole; no murmurs. No ankle edema. Red blood count, 3,500,000; hemoglobin 70 per cent. Urine slightly turbid; specific gravity, 1.012; albumin 4 plus; 30 white and 60 red blood cells per cubic millimeter; 2 large granular casts; nonprotein nitrogen, 56; chlorides, 425. The phenolsulfonphthalein test (P. S. P.) revealed 25 per cent excretion in one and one half hours.

Three weeks after admission, patient developed bronchopneumonia. Three days before death, nonprotein nitrogen, 83; creatinine, 2.9; carbon dioxide, 77.2; urea nitrogen, 68; chlorides, 440. On the last day patient became comatose with pulse 132, temperature 101.8° F.; abdomen markedly distended by ileus; marked pitting edema of both thighs.

Autopsy.—Generalized arteriosclerosis. Marked benign nephrosclerosis. Hypertrophy of the wall of the left ventricle. Anasarca of the lower extremities. Acute passive hyperemia of liver, stomach, spleen and kidneys. Acute hemorrhagic ulcerative colitis. Ileus. Bronchopneumonia.

Microscopic examination of the kidneys reveals advanced sclerosis of the intralobular and arcuate arteries rather than of the arterioles. There are disseminated regions where atrophy of convoluted tubules and groups of glomeruli with acute partial infarction and thrombosis are separated by fairly *well intact convoluted tubules and glomeruli.*

Summary.—A case of hypertension, arteriosclerosis with benign nephrosclerosis is presented. Death due to bronchopneumonia.

PLATE 55 HYPERTENSION WITH NEPHROSCLEROSIS AND ENDARTERITIS



Left ventricular hypertrophy (31 mm thick) Moderate number of small atheromatous plaques in the root of the aorta and aortic valve Coronary arteries unobstructed



The kidney surface is finely nodular The nodules are red purple and yellow tan On cut surface the medulla is dark purple The cortex is to 4 mm thick It is tan with disseminated red and purple points infarcted glomeruli due to occlusion of the small renal arteries

PLATE 55 HYPERTENSION WITH NEPHROSCLEROSIS AND ENDARTERITIS

I H, male, white, aged 58 Admitted on Nov 4 1939, died on Nov 22 1939

Patient had had hypertension known for three years and albuminuria of unknown duration Failing eyesight in the last two months A month before admission he had an attack of dizziness perspiration nausea and vomiting lasting for three days Admitted on account of hematuria of one week's duration, nocturia, scanty urination, marked dyspnea constant headaches, markedly impaired vision tinnitus nausea vomiting restlessness, insomnia, marked weakness Heart enlarged to the left, systolic murmur over precordium Blood pressure, 256/145 no ascites no edema of the extremities Urine bloody, specific gravity, 1 008 albumin 4 plus numerous red cells few white cells, few granular casts Hemoglobin 80 per cent, red cells 4 150, 000, white cells 12,650 Nonprotein nitrogen 56, carbon dioxide 78 2 volumes per cent chlorides, 390

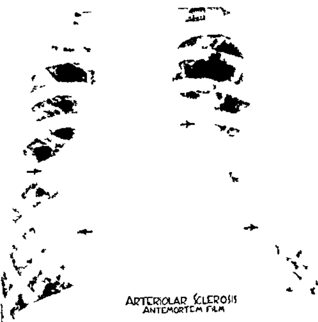
During the next two weeks, patient s condition remained unchanged elevated blood pressure, severe daily headaches nausea and vomiting, hematuria Five days before death patient had a generalized convulsive seizure, remained semiconscious respirations rapid and shallow, urinary incontinence developed and he became progressively worse

Autopsy —Generalized arterio and arteriolar sclerosis Nephrosclerosis Hypertrophy of the wall of the left ventricle 31 millimeters thick heart weighed 625 grams Myocardium firm and tan no areas of infarctions seen except for microscopic scars of the myocardium Acute passive hyperemia of the liver spleen stomach, and kidneys fatty changes of the liver Acute aspiration bronchopneumonia

Microscopic examination of the kidneys revealed a marked atrophy of the convoluted tubules and marked arteriolar sclerosis The walls especially the media of small arteries are tremendously thickened concentrically the increase in thickness due chiefly to hyalinized fibrous tissue occasionally the lumen cannot be identified A similar endarteritis with arteriolar sclerosis present in many other organs

Summary — A case of hypertension with nephrosclerosis and endarteritis is presented

PLATE 56 COR PULMONALE DUE TO PULMONARY HYPERTENSION AND ENDARTERITIS

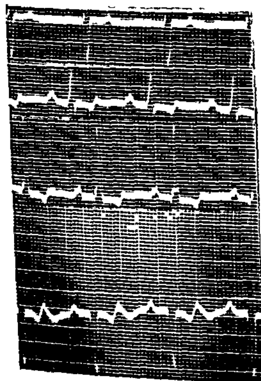


Bulging of left upper cardiac border due to dilated pul monary artery Increased hilai shadows due to dilated econdiry branches of the main pulmonary artery Heart enlarged to left and right due to right ventricular hyper trophy



Lungs Marked dilatation of the pulmonary artery and its branches Extensive arteriolar sclerosis and chronic endarteritis

S 6/41 Aged 40



Right ventricular strain Right axis devia tion with ST s depre ed T inverted



Marked hypertrophy of right ventricle and auricle due to ob truc tion in the pulmonary circulation by a chronic pulmonary endarteritis

PLATE 56 COR PULMONALE DUE TO PULMONARY HYPERTENSION AND ENDARTERITIS

J. G., male, white, aged 40. Admitted on July 30, 1941, died on Aug. 6, 1941.

Hemoptysis for two and one half months, cough and blood tinged sputum every morning. Dyspnea on slight exertion. Patient not acutely ill, but cyanotic. Heart and lungs negative. Blood pressure, 120/90. The liver palpable, about four fingerbreadths below the costal margin. Mild clubbing of the fingers, slight pitting edema of the feet. Temperature normal. Hemoglobin 14 Gm., red cells 4,500,000, white cells, 12,600. Kahn negative.

Two days before death, pulse 80, respirations quiet, marked venous pulsations in neck, lips cyanotic. Heart: apex beat forceful, almost in anterior axillary line; blowing systolic apical murmur. Blood pressure 120/80. Tentative diagnosis: cor pulmonale, probably due to progressive disease of the pulmonary arteries. Two days later patient very restless, quite cyanotic, pulse of poor quality, respirations shallow and rapid, 32 per minute. White count 21,600. The patient grew gradually worse and died that evening.

Autopsy.—Extensive arteriolar sclerosis and obliterative endarteritis and moderate atherosclerosis of the pulmonary arteries; dilatation of the pulmonary arteries; embolism thrombosis of the right pulmonary artery. Hypertrophy and dilatation of the walls of the right cardiac muscle and ventricle, engorgement of the cervical veins. Hydrothorax (right 50 cc.), hydropericardium (200 cc.), ascites (400 cc.), chronic passive hyperemia of the liver and kidneys. Atherosclerosis of the coronary arteries; fatty changes of the aorta. Clubbing of the fingers.

Summary.—A case of pulmonary endarteritis with hypertrophy and dilatation of the walls of the right cardiac chambers is presented with characteristic changes in roentgenogram and electrocardiogram.

The electrocardiogram of right ventricular strain indicates a marked right ventricular hypertrophy. The precordial electrode placed on the intersection of the left fifth interspace and midclavicular line normally would indicate changes, if any, in the apical portion of the left ventricle. Here, due to marked right ventricular hypertrophy, the precordial lead in the above location shows changes in the right ventricle similar to those in Leads 2 and 3. Compare with electrocardiogram and roentgenogram Plate 66.

PLATE 57 SYPHILITIC HEART DISEASE



Syphilitic aortitis aortic endocarditis and coronary arteritis Obliteration of the mouth of the right coronary artery with small scars in the posterior wall of the left ventricle Moderate hypertrophy of the left ventricle and chronic dilatation of the left ventricle and mitral ring Extensive right hydrothorax chronic passive hyperemia Generalized arteriosclerosis

M W male Negro aged 9 Chancre at age 19 treated for six months Wassermann and Kahn positive Dyspnea Orthopnea and swelling of the ankles for ten months Blowing systolic mitral and aortic murmurs Blood pressure 118/84 Right hydrothorax swollen liver edema of the ankles

V

SYPHILITIC HEART DISEASE

PLATE 57 SYPHILITIC HEART DISEASE

Syphilis of the heart and aorta affects the aortic valve, ostia of the coronary arteries, and proximal portion of the aorta.

The syphilitic aortitis involves the media of the aorta, causing perivascular *infiltration, inflammation*, necrosis, and later fibrosis, most marked in the proximal portion of the aorta. The fibrosis in the media soon extends to the intima and marked fibrosis and scarring, wrinkling, and pitting of the proximal end of the aorta is striking. Arteriosclerotic aortitis affects the intima forming atheromatous plaques, ulceration, and calcification, most marked in the distal portion of the aorta with greatest intensity in abdominal aorta.

The scarring and deformity of the proximal end of the aorta transforms it into a good resonant chamber and adds a musical bell or tambourlike quality *to the aortic second sound and as the scarring causes the aortic valve to close* under increased pressure the second sound is accentuated. In hypertension the increased blood pressure causes a snappy closure of the aortic valve and accentuation of the second aortic sound.

The syphilitic process in the proximal aorta soon spreads toward the aortic valve causing aortic insufficiency and to the mouths of the coronary arteries interfering with normal coronary circulation and causing coronary disease. Clinically syphilitic aortic insufficiency is similar to that of rheumatic or arteriosclerotic etiology. If the hypertrophied left ventricle fails the cardiac failure unrelieved may lead to death.

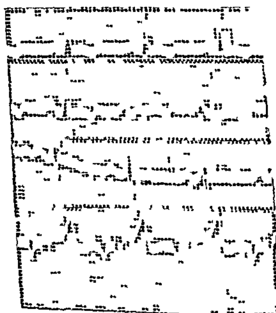
In arteriosclerosis the atheromatous plaques in the wall of the coronary arteries interfere with proper coronary blood supply and thrombosis in the lumen leads to myocardial infarction. In syphilitic coronary arteritis there is no thrombosis but a marked fibrosis and scarring of the mouths of the coronary arteries may lead to coronary occlusion and to myocardial infarction. Syphilis and arteriosclerosis frequently coincide.

PLATE 58 SYPHILITIC AORTIC INSUFFICIENCY, CORONARY DISEASE



Left ventricular border is rounded and out to the left due to left ventricular hypertrophy Note the interlobar fissure of the right lung

1 /15/43 Aged 5



Left axis deviation QRS inverted ST₁ elevated T inverted coved Coronary disease

PLATE 58 SYPHILITIC AORTIC INSUFFICIENCY, CORONARY DISEASE

L C, male, Negro Died suddenly at home at 54 years of age

Chancre at age of 16, syphilis untreated until twenty seven years later Wassermann and Kahn positive Development of cardiac symptoms—dyspnea on exertion, occasional nocturnal dyspnea due to left ventricular failure and dull precordial pain at age of 40 at which time the diagnosis of syphilitic aortic regurgitation was made Double aortic murmur loudest at the third interspace to the left of the sternum apical Austin Flint murmur Blood pressure 140 44 Corrigan pulse In addition to signs of aortic regurgitation (murmurs and pulse) patient had pulsating vessels of the neck chest arms and capillary pulse as evidence of associated vasodilatation

The electrocardiogram in 1932 was normal except for left axis deviation Electrocardiograms taken at various intervals from 1939 to 1943 indicate coronary disease with inverted T s The question of coronary disease being syphilitic with involvement of the mouths of coronaries or arteriosclerotic as expected at patient s age is of academic rather than practical value

Summary — A case of syphilitic aortic insufficiency with coronary disease as shown on the electrocardiogram is presented

PLATE 59 SYPHILITIC AORTIC ANEURYSM



Case 1 Ruptured aortic aneurysm



Case 2 Ruptured saccular aneurysm



View from bronchial side.



View from aortic side

Case 3 Saccular aneurysm of aortic arch with erosion into bronchus and fistula

PLATE 59 SYPHILITIC AORTIC ANEURYSM

A syphilitic aortitis may cause a localized dilatation and bulging, with a saccular aneurysm in the thoracic aorta Abnormal pulsation may be noted on inspection and a heaving pulsation on palpation over the upper anterior chest, with expansile quality, if the chest has been eroded Occasionally, an aneurysm fills with fibrin or thrombus and does not pulsate

Symptoms when present are due *to pressure on or erosion of adjacent* structures, as blood vessels (inequality of pulse and blood pressure in the two arms) nerves (inequality of pupils, hoarseness) trachea (tracheal tug) bronchus (cough), esophagus (dysphagia), spine (erosion), and ribs (expansile pulsation)

If the aneurysm compresses the innominate artery, the right radial pulse will be weaker than the left, while if the aneurysm lies between the innominate artery and the left subclavian the left radial pulse is delayed and weaker than the right Similarly, there may be differences in blood pressure in the two arms

Inequality of the pupils results from pressure on the sympathetics stimulation causing dilatation and paralysis producing contraction Hoarseness is due to pressure on the left recurrent laryngeal nerve and paralysis of the vocal cords

When the aneurysmal sac of the transverse arch of the aorta has become adherent to the trachea a tracheal tug may be elicited on palpation of the trachea *Pressure on the trachea may cause diminished breath sounds over* lungs In pressure on a main bronchus the breath sounds are diminished or absent over the affected lung

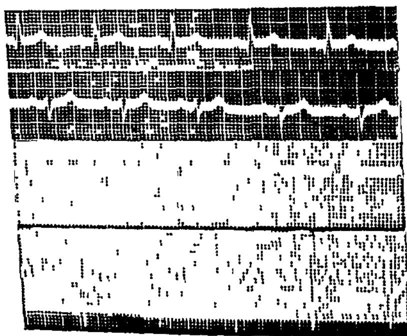
Aortic aneurysm may rupture and cause the patient's death

PLATE 60 SYPHILITIC AORTIC ANEURYSM



10/14/42 Syphilitic aneurysm of the aortic arch with pressure on left bronchus Trachea is displaced to the right

1/5/43 Aged 4[illegible] Rate 76



Left axis deviation

PLATE 60 SYPHILITIC AORTIC ANEURYSM

A V, white, male Died at 47 years of age

Chancre at 21 years of age with a short course of antisyphilitic treatment, but none after that Wassermann and Kahn positive Slight cough with expectoration of seven years' duration, dyspnea on exertion two years, no hoarseness An abnormal chest condition was found on pre employment physical examination

Pulse, 76, regular, equal in both wrists Blood pressure, 130/70 (left), 124/70 (right) Bulging and slight pulsation of the left upper chest, right chest is flat, prominent heaving apex impulse noticed to the left of the mid clavicular line, also noticeable suprasternal pulsation and bulging No mur murs Tracheal tug present in the neck Due to pressure of the aneurysm on the left bronchus, the left upper lung dull anteriorly and posteriorly, *breath sounds are distant, nearly absent no tubular breathing, no amphoric* sounds no râles cough and dyspnea

Patient died suddenly (rupture of the aneurysm?) at home on Oct 28, 1943 *No autopsy*

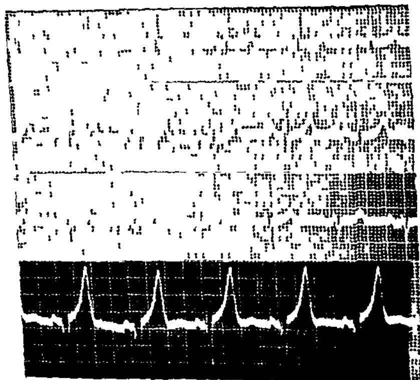
Summary—A case of syphilitic aortic aneurysm with pressure on the left bronchus is presented

PLATE 61 SYPHILITIC AORTIC ANEURYSM



1/16/43 Syphilitic aneurysm of the aortic arch with pressure and erosion of thoracic vertebrae

1/18/43 Aged 59



Left axis deviation

PLATE 61 SYPHILITIC AORTIC ANEURYSM

G P, white, male Died at 58 years of age

Chancre at 29 years of age, no treatment Twenty years later, dyspnea on slightest exertion Wassermann and Kahn positive Blood pressure, 140/60 Examination revealed syphilitic aneurysm of the aortic arch with aortitis, aortic insufficiency, and early cardiac failure Three years later (in 1940) dyspnea on exertion and severe pain in the back in the region of the thoracic spine Roentgenogram of the thoracic spine in 1943 revealed an erosion of the anterior and left borders of the fourth fifth and sixth thoracic vertebrae, secondary to a large aortic aneurysm On April 4 1944 we have following notation in our records "Patient coughs constantly, very short of breath *marked weakness, the pain in front of the chest and the back is worse and* extends downward into upper abdomen Heart to and fro murmurs at the base Lungs many moist and dry rales Tracheal tug Roentgenogram of the thoracic spine reveals further involvement and the bodies of two vertebrae are two thirds destroyed It is difficult to predict outcome but patient may have a sudden collapse at any time either from collapse of fourth fifth or sixth vertebrae and compression of cord (life is seldom possible when this occurs at this level) or rupture of the aneurysm " *Two weeks later patient* died suddenly at home No autopsy

Summary—A case of syphilitic aortic aneurysm with pressure and erosion of thoracic vertebrae is presented

practically reversing the normal relationship of the left and right heart as to the work performed

Defect in the interventricular septum is usually in the membranous, weakest part of the septum. The arterial blood from the left ventricle will flow into the right and mix with venous blood. There will be no cyanosis, and a systolic murmur in the third or fourth interspace to the left of the sternum may be the only evidence of septum defect. The same is true of acquired ventricular septum defect (Plate 40)

Pulmonary stenosis, if it occurs alone, without a ventricular septum defect, causes a systolic murmur in the pulmonary area, right ventricular hypertrophy, but no cyanosis, as there is no defect in the septum and no mixture of arterial and venous bloods will occur

In the tetralogy of Fallot, in addition to pulmonary valve, or in fundibular stenosis, there is interventricular septum defect, right ventricular hypertrophy, and dextroposition of the aorta. The tetralogy of Fallot is an example of venoarterial shunt, venous blood of the right ventricle mixing with arterial blood of the left causing a moderate to marked cyanosis. This kind of mixture is possible because of dextroposition of the aorta and ventricular septum defect with markedly increased pressure in the right ventricle over that of the left caused by pulmonary stenosis and hypertrophy of the right ventricle

The venoarterial shunt cyanosis should *not* be confused with cyanosis of right ventricular failure, peripheral stasis, or that of pulmonary origin, but in any cyanosis of whatever origin the abnormal color of the blood made visible by dilated capillary vessels is due to the increased amount of reduced hemoglobin

In venoarterial shunt a large number of red cells do not come in contact with oxygen in the alveoli of the lungs, and therefore there is insufficient oxygenation. As compensatory physiological adjustment, the oxygen capacity is increased by increasing the number of red cells, polycythemia, thus supplying sufficient oxygen to the tissues, with a result that the amount of oxygen in the blood is nearly normal

VI

CONGENITAL ANOMALIES OF THE HEART AND LARGE BLOOD VESSELS

Congenital anomalies are various defects in the structures of the heart and large blood vessels When an anomaly causes a cardiac failure, we may call it then a congenital heart disease Only a few of these anomalies which are compatible with life, and of clinical importance, are to be considered here

It is customary to include congenital dextrocardia in the discussion of congenital anomalies, although it is a normal heart, but congenitally transposed, with defect in the position and not in the structure of the heart

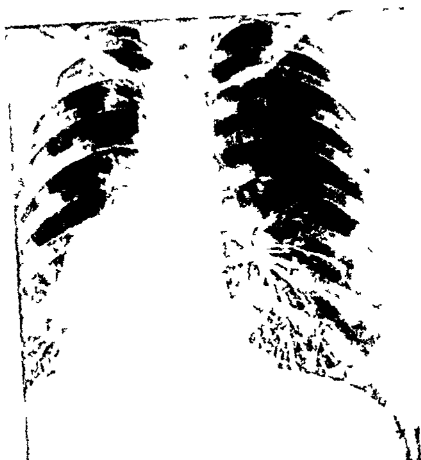
Congenital dextrocardia and coarctation of the aorta are examples of congenital anomalies without abnormal communication or shunt between the right and left side of the heart

Patent ductus arteriosus, auricular and ventricular septum defects, are examples of arteriovenous shunt with arterial blood entering the venous circulation

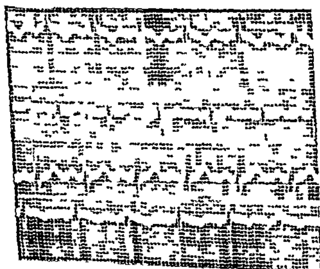
The auricular septum defects are (1) the patent foramen ovale, a frequently found anomaly and of no clinical importance (Plate 63), (2) persistent ostium primum (Plate 67), embryologically, the original opening in this part of the septum closes first, and (3) persistent ostium secundum, the original opening in the part of the septum in which foramen ovale is located, closes second, called also widely patent foramen ovale (Plate 66)

As the left auricle is situated anatomically above the right, the arterial blood of the left auricle in widely patent foramen ovale (Plate 66) enters the right auricle and mixes with its venous blood To take care of the extra load the right ventricle with tricuspid and pulmonic valves, the pulmonary artery and its branches will markedly hypertrophy The left ventricle and aorta will be small, hypoplastic, thus

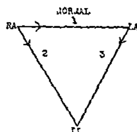
PLATE 62 CONGENITAL DEXTROCARDIA (TRANSPOSED HEART)



Cardiac apex and aortic knob on the right side Lipiodol visualizes transposition of the straight right and slant left bronchus



Lead 1 (P QRS T) invert d
Leads and 3 replace each other
Lead 4 inverted and similar to Lead 1

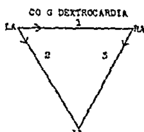


Lead —Normal

1 Right to left forearm
RA—→LA

Right arm to left leg
RA—→LL

3 Left arm to left leg
LA—→LL



Leads—
Congenital dextrocardia

1 LA—→RA
Lead 1 reversed

2 LA —→LL
Actually Lead 3 is taken

3 RA—→LL
Actually Lead 2 is taken

PLATE 62 CONGENITAL DEXTROCARDIA (TRANSPOSED HEART)

M M, female, white, aged 29, no cardiac complaints

Roentgenogram—Cardiac apex and aortic knob on the right side, as the heart is a mirror picture of a normally placed heart The gas in the stomach may be seen on the right side, as transposed heart is usually associated with transposition of abdominal organs The situs inversus involves also the bronchi and the lungs Lipiodol visualizes transposition of the straight right and the slant left bronchus See Plate 59, photograph of a trachea, straight right and slant left bronchus

Electrocardiogram shows characteristic changes due to transposition Leads 1 and 4 completely inverted, Leads 2 and 3 replace each other The electrocardiogram of the first three leads is similar to that which is found when lead wires of both arms are crossed, but Lead 4 in later instance is normal, not inverted

In acquired dextrocardia (see Plate 5) i e, right sided, displaced, and not transposed heart—as found in pneumothorax fluid, or adhesions—the apex aortic knob, and gas in the stomach are on the left side The electrocardiogram is normal

Dextroversio cordis (Plate 62) is an arrested development in early embryonic life when the heart did not complete its version or rotation from right to left, and remained on the right side The electrocardiogram is normal As dextroversio cordis is a developmental defect it is often associated with other congenital cardiac defects

PLATE 63 DEXTROVERSIO CORDIS, PULMONARY STENOSIS, SEPTUM DEFECTS

Marked stenosis of pulmonary valve Hypoplastic pulmonary artery Interventricular septal defect Right ventricular hypertrophy

Hypertrophy and dilatation of the aorta Closed ductus arteriosus Interventricular septal defect



Patent foramen ovale

PLATE 63 DEXTROVERSIO CORDIS, PULMONARY STENOSIS, SEPTUM DEFECTS

R M, male, white, aged 5½ Died on admission on March 8, 1943

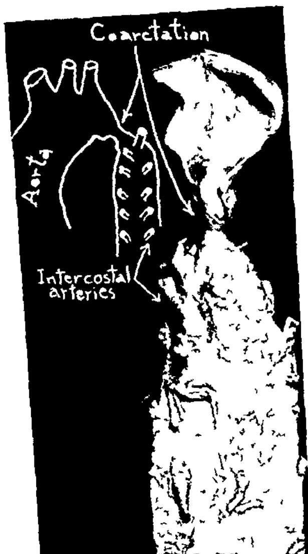
At one month of age, patient had cyanotic lips and diagnosis of dextrocardia was made The child had never walked, had started to talk at age 4, and was mentally retarded He was apparently well until one week before *admission*, when he developed measles, and then bronchopneumonia three to four days later Physical examination on admission revealed irregular, slow respirations, measles, cyanotic lips, feeble heart tones Patient died five minutes after admission

Autopsy—Marked stenosis of the pulmonary valve, with hypoplastic pulmonary artery (poststenotic hypoplasia), and hypertrophy of right ventricle and right auricle patent foramen ovale, interventricular septal defect, hypertrophy and dilatation of the aorta with closed ductus arteriosus Dextroversio cordis with the left side of the heart made of the left ventricle, the apex is that of the left ventricle and points to the right side Maculopapular rash of the head and trunk (history of recent measles) Hyperemia, edema, and disseminated hemorrhages of the lungs Acute mucopurulent bronchitis, acute and passive chronic hyperemia of the lungs liver, abdominal viscera, and brain Polycythemia and slight dehydration

Summary—A case of dextroversio cordis with pulmonary stenosis, interventricular septal defect and patent foramen ovale in a mentally retarded boy is presented Death due to bronchopneumonia

In dextroversio cordis the heart remains on the right side, due to failure of normal version of the heart from right to left The electrocardiogram is normal and does not show characteristic changes as found in congenital dextrocardia (transposed heart)

PLATE 64 COARCTATION OF THE AORTA



F K male aged 4 Died of bronchopneumonia

Constriction of the aorta at the site distal to the origin of the left subclavian artery and close to the insertion of the ligamentum arteriosum The constriction (coarctation) is sharp and annular—as constricted with a cord from outside while in stenosis the lumen is obstructed from within Note the dilated intercostal arteries arising from the thoracic aorta

PLATE 64 COARCTATION OF THE AORTA

The constriction of the aorta is distal to the origin of the left subclavian artery The blood pressure is increased in the preconstricted part of the aorta and arteries of both arms and decreased in postconstricted part of the aorta and arteries of the legs

A loud systolic murmur heard over the upper sternum and in the left interscapular region where the aortic arch is close to the spine

Collateral circulation is established through various arteries especially the intercostals which become prominent with visible pulsation The dilated intercostal arteries may cause erosion of the lower borders of the ribs as shown on the roentgenogram

PLATE 65 PATENT DUCTUS ARTERIOSUS

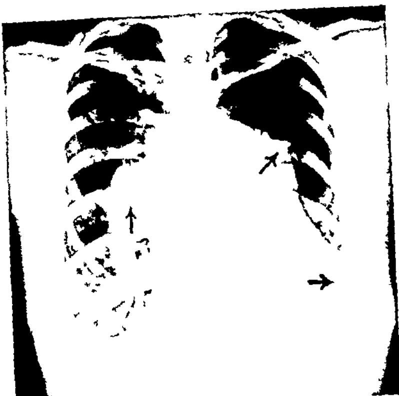


E L female white aged 36 Died of intra-abdominal hemorrhage following cesarean section The duct is between the left pulmonary artery (at a site close to the bifurcation of the main pulmonary artery) and aorta (at a site distal to the origin of the left subclavian artery) Note the glass rod in the duct The aorta is wide open and cut The pulmonary artery (sewed in here) is markedly dilated

PLATE 65 PATENT DUCTUS ARTERIOSUS

Patent ductus arteriosus is similar to arteriovenous aneurysm (see Plate 33) There is an abnormal communication between the arterial blood vessel the aorta and the venous the pulmonary artery The blood flows continuously as in arteriovenous aneurysm from the aorta into the pulmonary artery and causes a harsh loud continuous murmur, best heard in the second or third left interspace, or upper sternum and the left interscapular area where the aortic arch is close to the spine Due to increased blood volume the pulmonary artery is dilated and prominent on the roentgenogram

PLATE 66 WIDELY PATENT FORAMEN OVALE
AURICULAR SEPTUM DEFECT



Prominent pulmonary artery and conus The left lower cardiac border is out, rounded and globular due to hypertrophy of the right ventricle Increased hilar shadows due to dilated secondary branches of the pulmonary artery



Right ventricular strain right axis deviation with high wide QRS in all leads $ST_{2,3,4}$ markedly depressed deformed (slanting) and fused with deeply inverted $T_{3,4}$



Widely patent foramen ovale Chronic indurative mitral endocarditis Hypoplasia of left ventricle and aorta Note the marked hypertrophy of the right ventricle and the small size of the left



Marked hypertrophy of the right ventricle Hypertrophy of tricuspid pulmonic valves Hypertrophy and dilatation of pulmonary artery and its branches

PLATE 66 WIDELY PATENT FORAMEN OVALE AURICULAR SEPTUM DEFECT

A B, female, white, aged 38 Admitted on Feb 8 1942, died on Feb 19 1942

Pneumonia at 6 years of age, ill for about one month At 14 years of age, she was told she had leakage of the heart, which was not of recent origin or associated with infection at that time She was well until the second pregnancy at 25 years of age, when she first noticed dyspnea on exertion She had a cholecystectomy four years before admission At that time her heart revealed rough to and fro murmurs at apex and base A year later, dyspnea on *exertion set in with palpitation* at times but no edema On admission, the pulmonic sounds sharply accentuated and greater than aortic sounds a loud diastolic murmur heard over a large area of the upper sternum also tricuspid systolic and a loud mitral systolic murmur transmitted toward the axilla Blood pressure, 126/90

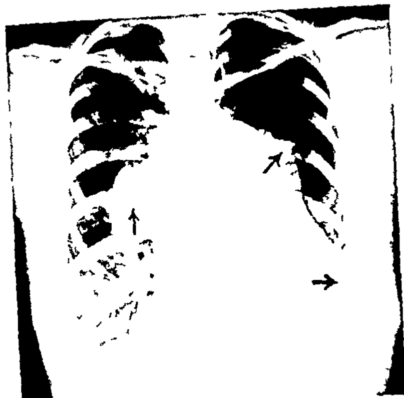
Exploratory thoracotomy for a suspected patent ductus arteriosus and possibly its ligation did not reveal any patent doctus Patient's temperature rose to 103° F and she died thirty six hours after the operation

Autopsy—Congenitally widely patent foramen ovale marked hypertrophy of the right ventricle 11 millimeters thick with work hypertrophy of tricuspid and pulmonic valves hypertrophy and dilatation of the walls of the pulmonary artery and its branches slight atherosclerosis of pulmonary artery Hypoplasia of left ventricle and aorta Chronic indurative mitral endocarditis Recent surgical defect of thoracic wall and incision of the pericardium acute serofibrinous pericarditis and left pleuritis compression atelectasis of left lung acute distention of right lung acute dilatation of right auricle Coronary arteries normal

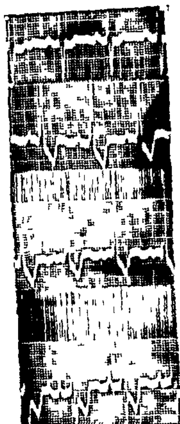
Summary — A case of widely patent foramen ovale with hypertrophy of the right ventricle and the pulmonary artery and its branches is presented Note the characteristic roentgenographic and electrocardiographic changes and compare with Plate 56 of cor pulmonale with pulmonary hypertension

The precordial electrode placed on the intersection of the left fifth interspace and midclavicular line normally would indicate changes if any in the apical portion of the left ventricle Here due to marked right ventricular hypertrophy the precordial lead in the above location shows changes in the right ventricle similar to those in Leads 2 and 3

PLATE 66 WIDELY PATENT FORAMEN OVALE AURICULAR SEPTUM DEFECT



Prominent pulmonary artery and conus The left lower cardiac border is out rounded and globular due to hypertrophy of the right ventricle In rea ed hilar shadows due to dilated econdary branches of the pul monary artery



Right ventricular strain right axis deviation with high wide QRS in all leads ST₂ ₃ ₄ markedly depressed de formed (slanting) and fu ed with deeply inverted T ₂ ₃



Widely patent foramen ovale Chronic indurative mitral endocarditis Hypoplasia of left ventricle and aorta *Note* the marked hypertrophy of the right ventricle and the small size of the left



Marked hypertrophy of the right ventricle Hyper trophy of tricuspid pulmonic valves Hypertrophy and dilatation of pulmonary artery and its branches

PLATE 67 AURICULAR SEPTUM DEFECT PERSISTENT OSTIUM PRIMUM

T B, female, white, aged 38 Admitted on Feb 14, 1942, died on Feb 15, 1942

Born prematurely (7 months) 2½ pounds at birth Normal child until 12 years of age, when she began to develop *scoliosis of the spine* *She was always* small In good health until eight months before admission, when she began to have dyspnea on slightest exertion and had to curtail her activities On admission pulse, 120 temperature, 99° F respirations 30 weight, 87 pounds blood pressure, 130/100 Small kyphoscoliotic white female Orthopnea and marked cyanosis Heart systolic blowing murmurs at the base and apex Fluoroscopy marked scoliosis with convexity to the right and rotation of the heart counterclockwise as in right oblique position, apex beat displaced to left pulmonary conus is prominent Clinical diagnosis marked kyphoscoliosis with cardiac failure due to torsion of the heart auricular septal defect with cyanosis tardive Patient expired day after admission

Autopsy—Congenital auricular septal defect (persistent ostium primum) with deformity of the mitral and tricuspid valves, induration of the mitral tricuspid, and aortic valves marked hypertrophy of the wall of the right ventricle dilatation of the right auricle Acute verrucous mitral and tricuspid endocarditis hydropericardium Hypertrophy and dilatation of pulmonary artery atherosclerosis of pulmonary artery Polycythemia Marked kyphoscoliosis of the spine Chronic passive hyperemia of the lungs liver and kidneys Compression atelectasis of lower lobe of left lung acute distention of right lung bronchopneumonia

Summary — A case of advanced heart failure due to marked kyphoscoliosis *of thoracic spine is presented* associated with congenital auricular septal defect and chronic induration of mitral tricuspid and aortic valves

PLATE 67 AURICULAR SEPTUM DEFECT PERSISTENT OSTIUM PRIMUM



Induration of mitral and tricuspid valves



Marked hypertrophy of the right ventricle Indurated tricuspid normal pulmonary valve



Induration of the aortic valve

PLATE 68 INFUNDIBULAR PULMONARY STENOSIS WITH INTERVENTRICULAR SEPTUM DEFECT

E O, male, white aged 22 Systolic thrill and loud systolic murmur at third and fourth interspaces to the left of the sternum Cyanosis dyspnea clubbing of the fingers and toes *Polycythemia (red cell count, 6 070 000)* with high hemoglobin (118 per cent) Patient died of pulmonary hemorrhage due to a far advanced pulmonary tuberculosis

Autopsy—The heart weighs 325 grams *The right ventricle (the wal* and the trabeculae) is much thickened up to 16 millimeters especially in its posterior and right lateral portions The main portion of the right ventricle communicates with the pulmonary conus (a conuslike part of the right ventricle situated below the pulmonary valve) through a 4 by 2 millimeter oval stenotic orifice The wall of the poststenotic conus is only 2 millimeters thick and does not participate in general hypertrophy of the right ventricle The pulmonary valve and artery are *normal* The right ventricle is also in communication with the left ventricle through a patency in the membranous portion of the interventricular septum The left ventricle is of normal thick ness 11 millimeters The mitral and aortic valves are normal The aorta is thin *walled and somewhat narrowed throughout* No dextroposition of the aorta as seen usually in Fallot tetralogy Ductus arteriosus is not patent

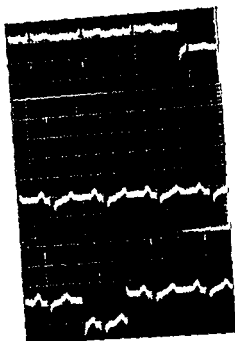
Summary—A case of infundibular pulmonary stenosis with interventric ular septum defect is presented The venoarterial shunt with cyanosis was due to two factors (1) the septum defect and (2) increased pressure of the right ventricle due to right ventricular hypertrophy so that the venous *blood of the right ventricle passed through* the septum defect and mixed with the arterial blood in the left ventricle

The stenosis is not in the valve itself but on the tip of the infundibular portion of the right ventricle (pulmonary conus) The systolic murmur in the infundibular pulmonary stenosis is heard best in the third or fourth interspace to the left of the sternum close to the location of the pulmonary conus

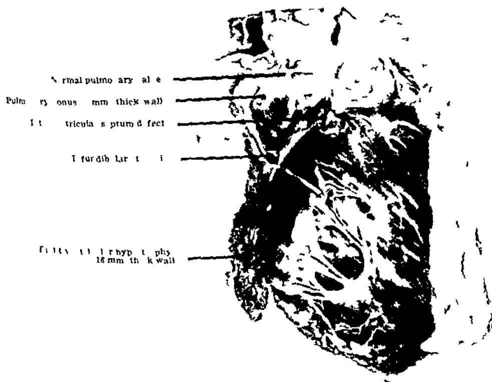
PLATE 68 INFUNDIBULAR PULMONARY STENOSIS WITH INTERVENTRICULAR SEPTUM DEFECT



Normal cardiac contour Pulmonary tuberculosis left lung



Right axis deviation due to right ventricular hypertrophy



Infundibular stenosis is on the tip of the pulmonary conus and not on the pulmonary valve Interventricular septum defect is in the membranous portion of the septum

PLATE 69 TETRALOGY OF FALLOT

J L , female, white aged 26 housemaid

Cyanosis since birth with dyspnea on exertion only Marked cyanosis of the skin, especially hands and feet, also ears nose, and lips Marked clubbing of the fingers and toes Vasodilatation and trophic changes with ulcers in the skin of both lower legs Heart a loud systolic murmur heard over entire precordium with greatest intensity in the third and fourth interspace near sternum, suggesting infundibular pulmonary stenosis Slight scoliosis of the thoracic spine with convexity to the left Erythrocytes 8,670 000, hemo globin 163 8 per cent, 27 3 Gm , color index, 1 0, leucocytes, 7,050

Roentgenogram—Heart is slightly globular in shape, probably due to hypertrophy of the right ventricle to the left and right The aortic knob is visible as increased round density on the right side indenting the barium filled esophagus to the left, while a normally placed aorta would indent it to the right The ascending aorta is on the left The roentgenographic evidence is a completely transposed aorta, while the dextroposition of the aorta in tetralogy of Fallot is usually a slight degree of transposition The pulmonary conus in infundibular pulmonary stenosis is the poststenotic portion of the right ventricle and is not visible on the roentgenogram because it is atrophic and hypoplastic

Discussion—Tetralogy of Fallot is a congenital anomaly with dextroposition of the aorta ventricular septum defect pulmonary (infundibular) stenosis, and right ventricular hypertrophy The marked cyanosis since birth indicates venoarterial shunt Red cell count is 8,670 000, hemoglobin, 163 8 per cent, 27 3 grams The shunt is about 50 per cent About one half of the venous blood of the right ventricle passes through the pulmonary artery into the pulmonary circulation The other half enters the aorta directly, due to dextroposition of the aorta and its origin from the right ventricle, and also passes through the ventricular septum into the left ventricle, due to increased right ventricular pressure (hypertrophy)

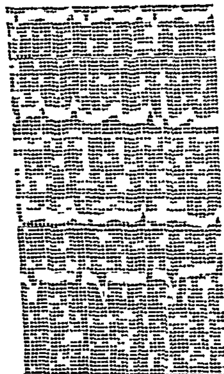
PLATE 69 TETRALOGY OF FALLOT



Marked cyanosis Clubbing of the fingers



Dextroposition of the aorta Indentation on a barium filled esophagus with convexity to the left



Right axis deviation P_2 high sharply peaked auricular hypertrophy T_4 normally inverted in children and occasionally in young adults

I NORMAL HEART

The greater portion of the left ventricle and both auricles are located posteriorly, and visualized on the roentgenogram in the left and right oblique views

In a normally built adult the heart is in an oblique position, while in a tall, thin person it is vertical, and in a stout, short individual it is horizontal In young children the heart is globular and centrally located

The electrocardiogram shows variations which are normal for that particular type of heart In the horizontal heart the electrocardiographic changes are in Lead 3 QRS3 low, notched, splintered, diphasic or inverted with T3 frequently inverted In the vertical heart the electrocardiographic changes are in Lead 1 QRS1 low, diphasic, or inverted In a globular heart a right axis deviation is found, which is normal in children

A normal heart is rotated by scoliosis of the spine In scoliosis with convexity of the spine to the right, the heart is rotated counterclockwise, and in the anterior view the heart contour is similar to that of the right oblique view In scoliosis with convexity of the spine to the left, the heart is rotated clockwise, and in the anterior view the heart contour is similar to that of the left oblique view

A normal heart may be displaced to the right or left, as by pneumothorax, fluid, or adhesions Except for a shifting axis, the electrocardiogram is normal in a rotated or displaced heart

II RHEUMATIC HEART VALVULITIS, MYOCARDITIS, PERICARDITIS, BACTERIAL ENDOCARDITIS

1 Mitral Stenosis

Mitral stenosis causes hypertrophy and dilatation of the left auricle and hypertrophy of the right ventricle The enlarged left auricle compresses the esophagus and left bronchus and while enlarging anteriorly and to the right pushes and rotates the heart counterclock

wise on its vertical axis, as shown on roentgenograms in anterior, left and right oblique views. The more marked is the stenosis, the more pronounced is the left auricular enlargement and rotation of the heart on the roentgenogram.

The electrocardiogram may show right axis deviation due to right ventricular hypertrophy and high, peaked P_2 due to auricular hypertrophy. Auriculitis in mitral stenosis may cause abnormal auricular myocardial conduction with deformed P_2 wide, notched, high or low, diphasic, or inverted. The anatomical changes in the left auricle may cause increased irritability and formation of auricular premature systole, paroxysmal auricular tachycardia (which is a succession of numerous auricular premature systoles), or, as frequently is the case, auricular fibrillation with abnormal initiation and conduction of auricular impulses.

2. Aortic Insufficiency

Aortic insufficiency causes hypertrophy of the left ventricle as shown on the roentgenogram in the anterior and left oblique views. The more marked is the insufficiency, the more pronounced is the left ventricular hypertrophy.

In mild cases, the left ventricular border, in the anterior view, from the end of the pulmonary contour down is rounded and extends below the diaphragm. In left oblique view the left ventricular border is rounded and heart is globular in shape. The electrocardiogram may show left axis deviation and high voltage of QRS.

In marked cases the left ventricular border is not only more rounded but extends out to the left in the anterior view and overlaps the spine in the left oblique view. At this stage a marked hypertrophy of the left ventricle may be followed by dilatation, with the left ventricular border markedly out to the left and overlapping the spine. The electrocardiogram shows left ventricular strain: left axis deviation with high wide slurred QRS, ST_1 depressed and fused with inverted T_1 and often inverted T_4. The left ventricular strain is an indication of a marked hypertrophy and not of a damaged myocardium. Dilatation and failure of the left ventricle with dyspnea may follow with signs of myocardial insufficiency in the electrocardiogram as tachycardia, low voltage of all leads.

If aortic insufficiency is associated with mitral stenosis, the enlarged left auricle may be seen in the right oblique view on the roentgenogram with compression of the esophagus in addition to the left ventricular hypertrophy of aortic insufficiency.

3 Pulmonary Stenosis (See Plate 18)

Pulmonary stenosis usually accompanied by mitral stenosis causes a marked hypertrophy of the right ventricle, especially the pulmonary conus i e the infundibular portion of the right ventricle situated below the pulmonary valve The hypertrophy of the right ventricle and pulmonary conus is more markedly pronounced than in the case of mitral stenosis alone The roentgenogram shows an enlarged left auricle on the right cardiac border and markedly counterclockwise rotated heart due to mitral stenosis In addition, a marked bulging and prominence of the pulmonary conus is seen, due to a marked infundibular hypertrophy and pulmonary stenosis, which cannot possibly be caused by mitral stenosis alone

The electrocardiogram will show right axis deviation and possibly auricular fibrillation as seen in marked mitral stenosis cases, but in addition right ventricular strain is present right axis deviation with $ST_{2,3}$ depressed, $T_{2,3}$ inverted, indicating a marked right ventricular hypertrophy

In aortic stenosis or marked aortic insufficiency there is a marked left ventricular hypertrophy as shown on the roentgenogram by marked changes on the left ventricular border and in the electrocardiogram by left ventricular strain In pulmonary stenosis there is a marked right ventricular hypertrophy as shown on the roentgenogram by marked bulging and prominence of the pulmonary conus of the right ventricle on the pulmonary mid left cardiac contour and in the electrocardiogram by right ventricular strain

4 Myocarditis

In myocarditis without impaired myocardial function the cardiac contour on the roentgenogram is normal Myocardial insufficiency and cardiac failure is associated with dilatation of cardiac chambers and the heart contour will be enlarged in all directions, affecting all the chambers of the heart This dilatation may be confused with pericarditis and at times differentiation may be difficult especially when myocarditis is associated with pericarditis Correlation with history physical findings, and the electrocardiogram is of great value in such cases

Myocarditis may cause impaired conduction in the myocardium proper or the auriculoventricular conduction system or both In contradistinction to myocardial infarction where the myocardial damage is usually localized, the rheumatic myocarditis is generalized of a disseminated type, causing changes in all the leads with disturbances in intraventricular conduction, as shown by wide, slurred, or notched splintered QRS deformed ST, or low, inverted T's An associated myocardial insufficiency may cause low voltage of all the leads and

tachycardia If the septum is involved, incomplete block with prolonged PR and dropped beats, complete block, or bundle branch block may be present

Serial electrocardiograms may reveal changes indicating activity of myocardial damage Usually, rheumatic myocarditis heals without any impairment of myocardial conduction and a normal electrocardiogram is found as indication of complete healing In some cases the scar tissue may interfere permanently with normal conduction Inverted T's or various forms of heart block remain permanently and unchanged for years as residue of an old healed damage

5 Pericarditis

The roentgenogram shows the cardiac shadow enlarged in all directions, gradually decreasing in size with absorption of the fluid

If pericarditis is associated with impaired myocardial conduction the electrocardiogram shows changes, as low voltage or inversion of all T's, which are valuable clues in doubtful or unsuspected cases of pericarditis In the majority of cases when recovery takes place, T's will be normal again, in others, inversion of all T's may remain permanently as a sign of a scar tissue in myocardium or pericardium, which interferes with a normal myocardial conduction and as such is of no clinical value

6 Bacterial Endocarditis

The roentgenogram and electrocardiogram reveal changes due to rheumatic heart disease but neither the roentgenogram nor the electrocardiogram are of much value as far as the bacterial endocarditis itself is concerned If cardiac failure sets in as a complication, it will manifest itself by enlargement of cardiac contour in all directions on the roentgenogram and low voltage of all leads with tachycardia in the electrocardiogram as indication of myocardial insufficiency

Hemorrhage, embolus, mycotic aneurysm with thrombosis or rupture in the vital organs of the body may be the cause of death in spite of successful treatment of the bacterial endocarditis, and sterilization of the blood stream with penicillin

III ARTERIOSCLEROTIC HEART DISEASE

Atherosclerotic process involves the aorta, mitral and aortic valves, and coronary arteries and may be associated with abnormal rhythm in the auricles, auricular flutter, and fibrillation

1 Atherosclerosis of the Aorta

The intima is altered by atheromatous plaques and calcification with increased severity from arch down toward the abdominal aorta

The weakened wall may dilate, causing a diffuse dilatation and tortuosity of the aorta, more frequently so when associated with hypertension Clinically, this condition is unnoticed by the patient and does not cause any symptoms, unless rupture of the weakened and dilated aortic wall occurs It may rupture completely and cause a fatal hemorrhage or may rupture partially with dissection of the coats of the vessel by blood (dissecting aneurysm)

If aorta is dilated and tortuous, the roentgenogram will show bulging *of the ascending aorta on the right upper cardiac contour*, prominent aortic knob, sometimes with calcification noted on the border, and the wide descending aorta will be seen as a diffuse shadow with a straight border extending from the aortic knob down and to the left of the pulmonary artery Frequently, the tortuous course of the thoracic aorta may be followed within the cardiac shadow down to the diaphragm The wide aortic arch and thoracic aorta can be better visualized in the left oblique view

Lateral view visualizes the wide abdominal aorta, especially when calcification is present

2 **Nodular Calcifying Aortic Stenosis**

Aortic stenosis causes a marked hypertrophy of the left ventricle The electrocardiogram of left ventricular strain and roentgenographic evidence of left ventricular hypertrophy are similar to that described in aortic insufficiency After reaching a maximum limit of hypertrophy, the left ventricle dilates and fails Unrelieved, the cardiac failure may be the cause of death

3 **Auricular Fibrillation**

As evidence of arteriosclerotic heart disease, the roentgenogram may show left ventricular hypertrophy or enlarged *heart in cardiac* failure, and the electrocardiogram may reveal other abnormalities besides auricular fibrillation

4 **Coronary Sclerosis and Myocardial Infarction**

The coronary sclerosis may be silent and inactive with slight changes in QRS, ST, and T in the electrocardiogram as the only evidence, if the conduction in ventricular myocardium is impaired, or various degree of heart block, incomplete, complete or bundle branch block may be present, if the septum is involved

The coronary sclerosis may be active The electrocardiogram, when compared with those taken previously, will show changes in various abnormalities in 2RS, ST and T found before

The myocardial infarction is usually found in two locations (1) in the apex anterior part of the left ventricle and adjoining anterior portion of the interventricular septum, so called anterior infarct with changes in the first and precordial leads, (2) in the base posterior part of the left ventricle and adjoining posterior portion of the interventricular septum, so called posterior infarct, with changes in second and third leads

In myocardial infarct the unaffected portion of the left ventricular myocardium may fail causing myocardial insufficiency with dyspnea, tachycardia, and low voltage of all leads in the electrocardiogram

The myocardial infarct may be the focus for cardiac irritability and cause appearance of premature systoles or paroxysmal auricular tachycardia, flutter or fibrillation

The localized necrosis in the myocardium leads to a localized involvement of endocardium with formation of a mural thrombus and of pericardium with pericarditis The thrombus may lead to embolus in systemic circulation and if a vital organ as the brain, is involved may be the direct cause of death while the myocardial infarction itself was clinically improving

The necrotic myocardium may rupture If the wall ruptures the hemorrhage may cause heart tamponade and death The rupture of the interventricular septum may heal without any ill effect leaving an opening in the septum

The active myocardial infarction may heal without leaving any clinical or electrocardiographic evidence of impairment In others the infarcted area causes localized thinning and bulging of the wall a ventricular aneurysm and impairment in myocardial conduction with permanent changes in QRS ST or T as residue of a healed infarct Ventricular aneurysm *with a well functioning* myocardium of the unaffected portion of the left ventricle may not cause any symptoms If the unaffected portion of the left ventricle fails, *myo*cardial insufficiency unrelieved may lead to death

IV HYPERTENSION

1 Systemic Hypertension

Hypertension in systemic circulation of any etiology causes hypertrophy of the left ventricle the same as in aortic insufficiency or stenosis The electrocardiogram shows left axis deviation or, in marked hypertrophy left ventricular strain Left ventricular failure may set in at this stage or after reaching a maximum limit of hypertrophy, the left ventricle dilates and fails Unrelieved, the left ventricular

failure with dyspnea is followed by that of the right, with cyanosis, peripheral edema, ascites, enlarged liver, hydrothorax, and finally death. Hypertension with left ventricular hypertrophy as shown on the roentgenogram and the electrocardiogram is not hypertensive heart disease. It may be called so, if cardiac failure is associated with and caused by hypertension.

2 Pulmonary Hypertension (Cor Pulmonale)

Increased pressure in pulmonary circulation as in pulmonary endarteritis (see Plate 56) causes hypertrophy of the right ventricle, cor pulmonale, and dilatation of the pulmonary artery and its branches. This is well shown on the roentgenogram: bulging of the left cardiac border in the region of the pulmonary artery, and increased hilar shadows due to dilated secondary branches of the pulmonary artery.

The right ventricular hypertrophy will cause right axis deviation and, if marked, right ventricular strain in the electrocardiogram.

The systemic hypertension causes hypertrophy of the left ventricle, while pulmonary hypertension that of the right. The condition can be recognized by changes on the roentgenogram. The electrocardiogram shows, left or right, axis deviation or ventricular strain.

V SYPHILITIC HEART DISEASE

The syphilitic process in the proximal aorta, aortitis, may spread toward the aortic valve, causing aortic insufficiency and left ventricular hypertrophy as shown on the roentgenogram and electrocardiogram. Aortitis may involve the mouths of the coronary arteries, causing coronary disease as shown in the electrocardiogram. The symptoms and electrocardiographic changes are similar to arteriosclerosis of coronary vessels.

A syphilitic aortitis may cause a localized dilatation and bulging, saccular aneurysm in the aortic arch which can be well shown on the roentgenogram.

VI CONGENITAL ANOMALIES

1 Congenital Dextrocardia

On the roentgenogram the cardiac apex and aortic knob are seen on the right side, as the heart is a mirror picture of a normally placed heart. The gas in the stomach may be seen also on the right side, as a transposed heart is usually associated with transposition of abdominal organs. The situs inversus involves the bronchi and the lungs. Lipiodol visualizes transposition of the straight right and the slant left bronchus.

The electrocardiogram shows changes due to transposition Leads 1 and 4 inverted, Leads 2 and 3 replace each other

2 **Coarctation of the Aorta**

The dilated intercostal arteries arising from the thoracic aorta may cause erosion of the ribs as shown on the roentgenogram

3 **Patent Ductus Arteriosus**

Due to increased blood volume the pulmonary artery is dilated and prominent on the roentgenogram

4 **Widely Patent Foramen Ovale Auricular Septum Defect**

As the left auricle is situated anatomically above the right the arterial blood of the left auricle in widely patent foramen ovale (see Plate 66) enters the right auricle and mixes with its venous blood To take care of the extra load the right ventricle and the pulmonary artery with its branches will markedly hypertrophy The roentgenogram is quite similar to that in cor pulmonale (see Plate 56) with exception of more outstanding findings of right ventricular hypertrophy In widely patent foramen ovale the right ventricle is exposed first to the increased pressure, while in cor pulmonale the pulmonary vessels are the first affected by increased pressure

The contour of a normal right ventricle in the anterior view (see Plate 1) is not visible The large central and anterior portion of the heart is the wall of the right ventricle The upper portion of it is the pulmonary conus of the right ventricle which lies medially to the pulmonary artery border The lower portion is the diaphragmatic surface to the left it is adjacent to the left ventricle and to the right is the right auricular appendage

In widely patent foramen ovale with marked hypertrophy of the right ventricle (see Plate 66) the pulmonary conus is bulging prominently on the left mid cardiac contour While the bulging may be due to a certain degree to dilated and hypertrophied pulmonary artery, the greater part of it is due to hypertrophy of the pulmonary conus, which overshadows the hypertrophied pulmonary artery The hypertrophy of the left portion of the right ventricle causes its rounding and enlargement to the left and when marked, overshadowing the normal straight left ventricular border The hypertrophy of the diaphragmatic surface adds to the rounding and enlargement to the left and also to the right by pushing the border of the right auricular appendage out Thus the heart is of globular shape with a marked bulging of the pulmonary conus added to it

The right ventricular hypertrophy causes right axis deviation or right ventricular strain in the electrocardiogram

5 Pulmonary Stenosis With or Without Ventricular Septum Defect

The congenital pulmonary stenosis may occur at the valve itself or on the tip of the infundibular portion, i e, the pulmonary conus of the right ventricle (see Plate 68)

In pulmonary valve stenosis, the pulmonary conus is markedly hypertrophied and bulges on the mid left cardiac border, similar to that in acquired type (see Plate 18)

In infundibular pulmonary stenosis, the pulmonary conus is the *poststenotic portion of the right ventricle and is not visible on the* roentgenogram because it is atrophic and hypoplastic

The right ventricular hypertrophy causes right axis deviation or right ventricular strain in the electrocardiogram

6 Tetralogy of Fallot

Tetralogy of Fallot is a congenital anomaly with dextro*position* of the aorta, ventricular septum defect, pulmonary stenosis and right *ventricular hypertrophy*

See description of Plate 69, also note dextroposition of the aorta on the roentgenogram and right axis deviation indicating right ventricular hypertrophy in the electrocardiogram

As only a part of the venous blood of the right ventricle passes through the pulmonary artery into *the* pulmonary *circulation* the insufficient oxygenation is compensated by polycythemia with increased number of red cells and increased hemoglobin, thus supplying sufficient oxygen to the tissues, with a result that the amount of oxygen in the blood is nearly normal

CLINICAL PATHOLOGY

The frequently encountered heart conditions can be divided etiologically into five groups rheumatic arteriosclerotic hypertensive syphilitic, and congenital Rheumatic and syphilitic are due to an infection, arteriosclerosis is a degenerative process of the arteries *while hypertension is an increased resistance* in the peripheral blood vessels due to various causative factors some unknown

I RHEUMATIC FEVER

1 Active Stage

Active rheumatic fever, affecting the heart causes active rheumatic heart disease Fever, leucocytosis and a high sedimentation rate are some of the general signs of activity while the appearance of a murmur, electrocardiographic changes pericardial rub are some of the local signs of activity in the heart Active rheumatic fever may

affect the endocardium with special predilection for valvulitis, mitral or aortic insufficiency, or both. The myocardium may be affected primarily or secondarily by extension from the endocardium. The inflammation may involve the ventricular wall or the interventricular septum or both. The electrocardiographic changes, as prolonged PR, dropped beats or complete auriculoventricular block suggest septum involvement. If present, it is a sign, which in correlation with other findings may lead to a correct diagnosis, but the absence of it does not exclude active rheumatic fever. The pericardium, the same as the myocardium may be affected primarily or secondarily from extension from the endo- and myocardium, causing carditis or pancarditis. Pericardial rub, roentgenograms and electrocardiographic changes, as low voltage, inversion of all T's, are some of the important diagnostic signs. The electrocardiographic abnormalities if present are valuable clues in doubtful or unsuspected cases of pericarditis.

2 Inactive or Healed Stage

The stage of activity and healing is followed by an inactive stage of a healed process with anatomical and physiological adjustments. Following mitral insufficiency, a fibrosis of the valve causes a mitral stenosis which is an indication of a healed condition, with a moderate or marked hypertrophy of the left auricle and right ventricle to compensate it. In aortic insufficiency left ventricular hypertrophy sets in early, as well as physiological adjustment of the blood pressure. In myocarditis and pericarditis electrocardiographic changes may completely disappear or if the scar tissue and fibrosis interfere with conduction permanent electrocardiographic change may remain as residue of a healed condition.

At this stage a murmur as evidence of a valvular lesion, auricular or ventricular hypertrophy, electrocardiographic and roentgenographic change are signs of an inactive, healed and well compensated process, and the patient does not need any treatment for his condition.

3 Cardiac Failure, Stage of Breakdown

If for any reason a breakdown of compensation occurs in inactive rheumatic heart disease a *cardiac* failure with failure of the function of the ventricular myocardium sets in and the failing heart must be helped by the physician to carry on the required physiological processes or a complete breakdown will lead to death.

At this stage of breakdown of compensation the physician must evaluate the extent of anatomical and physiological impairments in the heart, he must correlate the electrocardiographic and roentgenographic findings as for instance, the extent of auricular or ventricular

hypertrophy with clinical signs of left or right ventricular failures The knowledge of the degree of cardiac damage and its adjustment by the heart is most helpful to the physician as to the proper dose of the medication to be used

4 Bacterial Endocarditis

Bacterial endocarditis is a complication of inactive rheumatic heart disease In addition to septic fever, enlarged spleen, positive blood culture, and cardiac murmur, the bacterial endocarditis manifests itself by various vascular phenomena (1) emboli, as cerebral or Osler's nodes on fingers and toes, (2) arteritis, with thrombosis and mycotic aneurysm, as of cerebral or coronary blood vessels, (3) hemorrhages, as cerebral, renal, or petechiae If these manifestations affect the vital organs, they may cause death in spite of successful treatment of the bacterial endocarditis and sterilization of the blood stream with penicillin The same is true, when bacterial endocarditis is complicated by cardiac failure

II ARTERIOSCLEROSIS

Arteriosclerosis is not a disease but a degenerative process involving the peripheral artèries

1 Atherosclerosis of the Heart and Aorta

Atherosclerosis of the heart and aorta involves the aorta, mitral and aortic valves, coronary arteries, and may be associated with abnormal rhythm in the auricles, auricular flutter, or fibrillation In the *atherosclerosis of the aorta*, the intima is altered by atheromatous plaques and calcification, with increased severity from the arch down toward the abdominal aorta The weakened wall may dilate, causing a *diffuse dilatation and tortuosity* of the aorta more often so when associated with hypertension Clinically, this condition is unnoticed by the patient and does not cause any symptoms, unless rupture of the weakened aortic wall occurs It may rupture completely and cause a fatal hemorrhage or may rupture partially with dissection of the coats of the vessel by blood In dissecting aneurysm, death may not occur immediately, but may be postponed for days, weeks, or even months

The atherosclerosis of the aortic valve may cause aortic insufficiency, with *a soft diastolic aortic murmur, characteristic blood pressure* changes and left ventricular hypertrophy This condition if followed or complicated by left ventricular myocardial (cardiac) failure *may be called then* arteriosclerotic heart disease

Fibrosis and sclerosis may cause slight aortic stenosis, with soft systolic aortic murmur or nodular calcifying aortic stenosis with pro

nounced blowing systolic aortic murmur, small plateau pulse, normal or slightly lower blood pressure and marked hypertrophy of the left ventricle Here again, if this condition is followed or complicated by left ventricular myocardial failure, it may be then called arteriosclerotic heart disease

2 Coronary Sclerosis and Myocardial Infarction

Atherosclerosis of the intima of the coronary arteries is a degenerative process similar to that in any other arteries The coronary sclerosis may be silent and inactive with slight changes in QRS, ST, and T as the only evidence, if conduction in ventricular myocardium is impaired, or various degrees of heart block incomplete, complete, or bundle branch block may be present, if the septum is involved

Thrombosis and occlusion of the lumen of a branch of a coronary artery with a resulting localized myocardial infarction and necrosis is a complication and medical accident due to arteriosclerosis of coronary arteries This is most frequently encountered in two locations (1) in the apex, anterior part of the left ventricle and adjoining portion of the ventricular septum, so called anterior infarct, a part supplied by anterior descending branch of the left coronary artery and (2) in the base, posterior part of the left ventricle and adjoining posterior portion of the ventricular septum so called posterior infarct a part supplied by the circumflex branch of the left coronary, or the right coronary artery In myocardial infarct, especially when extensive the unaffected portion of the left ventricular myocardium may fail the myocardial insufficiency in this case being the result of a marked coronary insufficiency Clinically, there will be dyspnea as a sign of left ventricular failure In some cases the myocardial infarct may be the focus for cardiac irritability and cause appearance of premature systoles or paroxysmal auricular tachycardia, flutter, or fibrillation The localized necrosis in myocardium leads to a localized involvement of endocardium with formation of a mural thrombus and of pericardium with pericarditis The thrombus may lead to embolism in systemic circulation, and if a vital organ as the brain is involved may be the direct cause of death, while the myocardial infarction itself was clinically improving The necrotic myocardium may rupture If the wall ruptures, the hemorrhage causes heart tamponade and death The rupture of the ventricular septum may heal without any ill effect leaving a small opening in the muscular portion of the septum

The active myocardial infarction may heal without leaving any clinical or electrocardiographic evidence of impairment In the others the infarcted area causes localized thinning and bulging of the wall ,

ventricular aneurysm, and impairment in myocardial conduction with *permanent changes in the electrocardiogram* Clinically, there may be dyspnea as a sign of left ventricular failure

The episode of myocardial infarction may be followed later by another in the same or another area, with consequences as already stated

III HYPERTENSION

1 Systemic Hypertension

Increased tension in peripheral arteries is an abnormal finding and not a disease To overcome the high peripheral resistance, i e, the higher diastolic pressure, the power of the heart, i e, systolic pressure increases with resulting hypertrophy of the left ventricle When dilatation of the left ventricle sets in with myocardial insufficiency, *dyspnea and left cardiac failure as physiological and clinical evidence* of it, we are dealing then with hypertensive heart disease If unrelieved, the left ventricular failure with dyspnea will be followed by that of the right with cyanosis, peripheral edema, ascites, enlarged liver, hydrothorax, and finally death

2 Pulmonary Hypertension

Any obstruction in pulmonary circulation as *endarteritis of the* pulmonary arteries causes pulmonary hypertension with hypertrophy of the right ventricle, so called cor pulmonale The right ventricle may fail and cause death

IV SYPHILIS

Syphilis of the heart affects the proximal portion of the aorta, the aortic valve and the ostia of the coronary arteries Clinically, syphilitic aortic insufficiency is similar to that of rheumatic or arteriosclerotic etiology If the hypertrophied left ventricle fails, the cardiac failure unrelieved, may lead to death The occlusion of the mouths of the coronary arteries may interfere with normal coronary circulation and cause coronary heart disease Syphilis and arteriosclerosis of the heart frequently coincide

A syphilitic aortitis may cause a localized dilatation and bulging, a saccular aneurysm in the aorta Symptoms when present are due to pressure on or erosion of adjacent structures

Aortic aneurysm may rupture and cause the patient's death

V CONGENITAL ANOMALIES

Congenital anomalies are various defects in the structures of the heart and large blood vessels When an anomaly causes a cardiac failure it may be called then a congenital heart disease

Coarctation of the aorta is an example of congenital anomaly without abnormal communication or shunt between the right and left side of the heart Patent ductus arteriosus, auricular and ventricular septum defects are examples of arteriovenous shunt with arterial blood entering the venous circulation In tetralogy of Fallot, in addition to pulmonary stenosis there are ventricular septum defect dextroposition of the aorta and right ventricular hypertrophy Tetralogy of Fallot is an example of venoarterial shunt, venous blood of the right ventricle mixing with arterial blood of the left causing cyanosis

This kind of mixture is possible because of dextroposition of the aorta and venous blood of the right ventricle entering the aorta directly while increased right ventricular pressure due to right ventricular hypertrophy permits the venous blood of the right ventricle to enter the left ventricle through the ventricular septum defect Polycythemia is a compensatory adjustment mechanism for the insufficient oxygenation of the blood

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